# Silicosis and Asbestosis

BY VARIOUS AUTHORS

EDITED BY

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OXFORD UNIVERSITY PRESS

LONDON NEW YORK TORONTO

*Printed in the United States of America*

# PREFACE

THE industrial age has complicated the scheme of life in many ways, some of them directly affecting the public health; among these are the diseases due to the inhalation of certain kinds of atmospheric dusts arising directly from industrial operations Ill effects due to dust contamination of the atmosphere have been recognized from earliest times.

This book has for its purpose the presentation of the medical and public health aspects of silicosis and asbestosis, two definitely industrial dust diseases Silicosis looms larger in the picture than asbestosis because the former is much more widely spread, affects a much greater number of the industrial population, has been recognized and studied longer and, consequently, more is known about it

The form of the generic term pneumoconiosis, rather than pneumonoconiosis or pneumonokoniosis, has been adhered to throughout the book The International Labour Office and both British and American editors have adopted it as preferable

The editor wishes to express special thanks to Miss C M Bresnan for reading proof and for making the index and to acknowledge with gratitude the help received from many sources and from many individuals To them and particularly those working in the field of industrial hygiene, this book is dedicated

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*February* 24, 1938

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## CONTENTS

ILLUSTRATIONS

PAGE

CHAPTER IV

CHAPTER V

CHAPTER VI

# INTRODUCTION

PATHOLOGICAL conditions of the lungs, due to the inhalation of excessive quantities of dust in certain occupations, have been known to exist for many centuries It is also true that the frequency of infection, particularly pulmonary tuberculosis, as a complication has been realized for a long time Pneumoconiosis is a comprehensive term, designating *the effects upon the lungs of the inhalation of excessive quantities of dust,* manifested by structural changes in the lung tissue and entirely distinct from the action of poisonous dust, such as lead or mercury, in which case the lungs act *merely as the point of entrance into the body without definite local influence.* At first it was associated chiefly with mining but with the advancement of the industrial age, dusty industries multiplied.

It is customary to speak of the dust inhalation effect upon the lung tissue by the name of the chief constituent of the dust breathed, as silicosis when silicon dioxide ($SiO_2$) is the principal ingredient and asbestosis when asbestos is the offending agent Silica dust, when the particles are sufficiently small, has the peculiar property of inducing a fibrosis of the lung tissue This effect is characteristic, as is the resulting roentgenologic appearance. In asbestosis, the pathological process is quite different as is the roentgenologic appearance These matters are thoroughly dealt with in the text. There is still, however, much to be learned with respect to both conditions We know the results of the inhalation of these dusts but we are not sure how they are produced.

There are two ways in which the pulmonary infections, tuberculosis and pneumonia, may affect or complicate pneumoconiosis First, there may be infection before the exposure

to dust. It is an undecided question whether such infection can so change the pulmonary structure that the later dust effect is more pronounced than it would otherwise be. There seems to be reason to believe that such an enhancement is to be expected, especially if the previous infection were tuberculosis, but this must be left to the histopathologist for final proof. Secondly, do such infections upon an already dusted lung become more severe or aggravate the dust effect or otherwise change its character? There is even more evidence that any of these effects may result but here again the histopathologist must be called upon to tell us the exact truth The first intimation that occurred to us that the character of a silicotic lesion could be so changed was when we were searching for an explanation as to why certain individuals with silicosis showed massive consolidations of fibrosis in the lungs and others in the same occupation and apparently exposed to the same amount of dust of the same variety showed a more evenly diffused nodular fibrotic process It was a great satisfaction to hear a well known pathologist say that a comparatively recent infection like lobar pneumonia might be the answer but that at present there was no definite proof of such theory being correct

It was gratifying to learn that most of us were thinking along similar lines That most cases of silicosis die of an intercurrent infection, especially tuberculosis and possibly pneumonia, is evidence that silicosis may exert some effect upon the progress of some pulmonary infections It has not been proven as yet that the same is true of asbestosis.

A comprehensive knowledge of the effects upon the lungs of excessive inhalations of dust is not to be quickly gained The combined efforts of the histologist, pathologist, clinician, roentgenologist chemist, physicist and other technological experts are necessary. The legal profession deserves a certain share of commendation for stimulating the study of

occupational dust diseases and has made a thorough knowledge of pneumoconiosis a necessity among many of the medical men Were it not for the demand for exactness from outside the medical profession, it is difficult to say how soon curiosity or medical research would have achieved our present accuracy of knowledge with respect to the etiology, symptoms, and pathology of pulmonary dust diseases and their modification by certain infections

A large number of us have, no doubt, taken up the study of pneumoconiosis from the standpoint of interest Following the experience with silicosis in South Africa, starting several decades ago, text books for the medical student simply mentioned it in a more or less cursory manner as an uncommon condition We are sure of the necessity of many angles of approach to the subject The first comprehensive report of silicosis in the United States was made about twenty years ago by one of the authors. It described what was occurring among miners in the south west Almost simultaneously and without any knowledge of this report, it occurred to a small group of medical men in the east that the best way to study the differences in the appearances of trunk shadows in the roentgenograms of the lungs might be to study the chests of persons engaged in various dusty occupations The main point learned in this investigation was that organic dusts do not induce lung changes but it further stimulated the study of the effects of inorganic dusts, particularly silica, and later, asbestos This study has continued up to the present time.

Some of the authors of this book were among the pioneers in silicosis and asbestosis; they have blazed the trail for future investigations It can be truthfully stated that those who studied these occupational diseases in North America and the British Empire have always had the good of the affected workers in mind. Their researches have saved life and have made many occupations safer for the worker

HENRY K. PANCOAST, M D.

SILICOSIS
AND
ASBESTOSIS

# I. HISTORY OF SILICOSIS AND ASBESTOSIS

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## 1 HISTORY OF SILICOSIS

ALTHOUGH to the general public silicosis is a disease of recent origin, the history of dust diseases reaches far into the past, and the first recognition of a definite relation between exposure to dust and pulmonary infection is lost in the mists of antiquity

As Collis[1] states, 'In historic times references are to be found in the works of ancient writers who, however, seldom distinguish between the various forms of respiratory disease, but rather refer to a general connection between lung affections and dust inhalation'

Probably the earliest recorded reference to the harmfulness of dust exposure is Pliny's description of the devices used by refiners to prevent inhalation of the 'fatal dust'[2] The symptoms of a metal digger, as noted by Hippocrates, who lived in the fifth century B C, are comparable to those observed among present-day miners suffering from the effects of dust inhalation[3] Other early writers discussed dust phthisis, and it is believed they had in mind the type of disease described by Celsus,[4] a medical writer of the first century, who stated

'By far the most terrible form of emaciation is that which the Greeks call phthisis It spreads to the lungs On top of this ulceration occurs and a slow fever which at times disappears and at other times reappears'

In 'De Re Mettalica,' published in 1556, Agricola described the perils of mining and the 'pestilential air' breathed by miners He also relates how 'the constant dust enters the blood and lungs, producing that difficulty of breathing the Greeks call asthma When the dust is corrosive it ulcerates the lungs and produces consumption.' 5

Lung diseases of miners, smelters, grain workers, etc., were discussed and described by a number of early writers. Among these, Paracelsus, a Swiss alchemist and physician, was the first to list the occupational diseases of miners and smelters. His book on industrial medicine was published in 1567.[6] It is said that Dimerbroek, in 1649, made the first section of a stone-cutter's lung which revealed 'lung vesicles completely clogged with fine dust' 7 Ramazzini, in his book published in 1700, noted the effect of dust on the respiratory organs, and reported numerous cases of fatal dust disease. He obtained his information by personal inspection of the trades he discussed, and his writings display a recognition of the relationship between dust inhalation and consumption 8 According to Collis, Thomas Benson, of Newcastle-under-Lyme, was granted a patent for grinding flints by a wet method. Previously, flints were pounded dry, the process proving 'very destructive to mankind insomuch that any person, ever so healthful and strong, working in that business, cannot possibly survive over two years, occasioned by the dust sucked into his body by the air he breathes' 1 A number of special inquiries into the prevalence of dust-phthisis were made, for example, by Professor Allison 9 and Hugh Miller,[10] who wrote of the progress of such disease among stone-cutters

Thackrah,[11] in 1831, began a study of the effect of the dusty trades on British workmen's longevity, and contributed valuable information, including the fact that not all dusts shorten the lives of the workers exposed, as evidenced by the experience in various mines He noted that, while sandstone workers generally died before forty, there seemed to be little or no unusual incidence of lung diseases in brick and limestone workers

Dr Greenhow,[12] an instructor in public health, became the first state medical factory inspector in England and made personal investigations of health conditions in industries throughout the country He collected lung specimens of workers, who had died from dust diseases, for pathological study and he was perhaps the first to use polarized light to identify microscopically the tiny particles of silica in the lungs of a metal grinder As a result of his work, the first Royal Commission was appointed in 1861 to make a thorough investigation of health conditions in mines.

In 1862, Dr Peacock,[13] who had interested himself in the lung diseases of buhrstone cutters and who had detected particles of the stone in their lungs, was made a member of the Commission In his report on the medical aspects of mining, he distinguished between the conditions of the lungs known as 'miner's phthisis' and the ordinary type of tuberculosis, noting that the miner's disease occurred in otherwise healthy men with healthy families and that it appeared in them at an older age and was a more chronic type than the usual infective tuberculosis

Although medical opinion at this period attributed the disease among miners more to bad hygienic conditions than to the dust to which they were exposed, some of the Scottish and English physicians noticed that even in mines where miner's phthisis was prevalent, drillers of rock suffered from more rapid onset of the disease than other miners This, to-

gether with other similar data, gave rise to the supposition that the character of the rock influenced the occurrence of the disease.[14]

The effects of various dusts on the lungs were reported by other writers, the names given to the resulting diseases being descriptive of the dusts causing them. Zenker[15] provided the anatomical and Kussmaul,[15] the chemical proof of the deposit of inhaled dust, especially siliceous dust in the lungs. The name 'pneumonokoniosis' was devised by Zenker, who described in detail the anatomical picture of the dusty lung.

Visconti, as reported by Rovida,[16] in 1870, is accredited with being the first to use the term 'silicosis' to denote the pathological condition of the lungs resulting from inhalation of silica.

Commencing in the latter part of the nineteenth century, great interest in the health of miners was being shown throughout the world A summary of development in some of the principal countries follows.

## South Africa

Too much credit cannot be given to the South African authorities and investigators for their contributions to the whole subject of pulmonary dust diseases The publications and reports of the Miner's Phthisis Prevention Committee, the Miner's Phthisis Medical Bureau, and the South African Institute for Medical Research have been an inspiration for officials and research workers all over the world Their influence on both the clinical and compensation aspects of silicosis has been enormous

Gold mining started in South Africa on the Witwatersrand in 1886 It was not long before the introduction of machine drilling and the depths of the mines began to produce health problems The quartz content of the ore bearing rock was high, containing as much as 80 per cent to 90 per cent free

silica In 1902, the Government mining engineer noted that miner's phthisis seemed to be prevalent among rock drillers and a Commission of Enquiry was appointed. Physical examinations were made of 3,000 men, the roentgen ray being used in South Africa for the first time in 300 cases.

Compensation was inaugurated in 1912 and in 1916, the Miner's Phthisis Prevention Committee issued a comprehensive report Pre-employment and periodic physical examinations were initiated by the Medical Bureau and engineering methods to improve ventilation and allay dust by the use of water were instituted The estimation of the amount of silica dust in various working places underground was developed, Sir Robert Kotze having devised an instrument for this purpose, the Kotze Konimeter A threshold limit of 300 particles per cubic centimeter (about 8,500,000 particles per cubic foot) was set as a reasonable (at that time) standard of safe practice

The Miner's Phthisis Medical Bureau defined diagnostic standards and regulations for compensation The Bureau established the classification of silicosis into four stages — ante-primary, primary, secondary, and silicosis with tuberculosis Watkins-Pitchford and the other medical members of the Bureau — Watt, Irvine, and Stewart — made notable contributions to clinical knowledge The importance of tuberculous infection as an accelerator of lung fibrosis, the marked susceptibility of silicotics to tuberculosis, the progressive nature of silicosis were among the important aspects of silicosis which this group described From the Institute for Medical Research came the important pathological studies of Mavrogordato and Simson. No one interested in the medical, economic, or engineering phases of silicosis can afford to overlook the publications that have come from South Africa for the past twenty years

## Great Britain

In 1902, fifty years after Peacock's investigations, a governmental commission was appointed to study the persisting high mortality among the tin miners of Cornwall. Dr. Haldane, one of the members of this commission, definitely ascribed the disease of these miners to rock dust and stated that the dust itself was what caused the primary injury to the lungs.[17]

Investigations among slate workers showed that slate dust exposure was related to phthisis, particularly in Wales, where the slate is high in quartz.[18]

From this time on, frequent and intensive investigations of many of the dusty trades in England were made, and numerous reports containing valuable information and suggestions were issued by the British Home Office Among the industries studied were slate, refractories, sandstone, potteries, and coal mining

Dr Collis, in 1915, in the Milroy Lectures, reviewed the status of pneumoconiosis in England at that time. He clearly expressed the theory that 'free' or crystalline silica is the causative agent in nearly all dusts that produce serious lung injury and susceptibility to tuberculosis [1]

In 1917, an investigation was made of the refractories industry, where a species of sandstone shale with a high free silica content is used in making bricks and in 1919, the first scheme of compensation for silicosis was set up for this industry. Dr. Heffernan has given an account of the problems connected with this industry [19]

The situation in the English pottery industry had existed for many years, Wedgewood having found that his workers employed in grinding flint lived only a couple of years Several Royal Commissions were appointed to investigate the potteries industry; one by Sutherland and Bryson for-

mulated a scheme for including the potteries in compensation laws.[20] One report of the British Factory Inspectors states that regulations now enforced for controlling dust in this industry has been so effective that no process in English potteries is now so hazardous as to produce even an early stage of silicosis in less than ten years.

The granite industry was also studied repeatedly. In 1930, out of about 500 workers examined, 17 per cent showed evidence by roentgen-ray of silicosis, mostly in the early stages. The free silica content of granite is lower than that of sandstone, usually not over 30 per cent, but the fact that pneumatic tools were generally used was thought to cause more dust than hand methods.[21]

A very comprehensive report on the metal grinding and casting cleaning industry was issued by two of the Factory Inspectors in 1923. It was shown that the occurrence of silicosis, many cases being complicated with tuberculosis, was present almost entirely among those who worked on sandstone grinding wheels. Silicosis was more frequent and more advanced in the wet grinders examined than in the dry grinders. Sandblasting was rated as potentially a very hazardous process, unless enclosed, or the workers protected. Counts of the dust particles in the air were made with an apparatus developed in 1922, called the Owens' jet dust counter. Petrographic determinations were made and particle size measurements estimated for the dust present in the various processes. Detailed requirements were drawn up for the improvement of working conditions. This report helped to establish the fact that metal particles or hard mineral particles from corundum or emery were not the source of true silicosis; that only work on sandstone wheels or on sand cleaning or with other exposure to free silica dust is capable of producing the disease.[22]

Collis and Yule, in 1933, endeavored to determine the ef-

fects of exposure to silica dust on mortality by comparing the death rates for a group of occupations having exposure to dust containing free silica with a group having equal exposure to dusts not containing free silica, working conditions being similar Comparison was also made with the rates for all occupied and retired males, with correction for age distribution The authors concluded from this analysis that silica dust tends to be a poison to the whole body and that, although it exerts its important influence on the respiratory tract, especially with respect to tuberculosis, it also impairs the circulatory, nervous, and digestive systems and the glands, kidneys, and liver [23]

The chemical aspects of the action of silica in the tissues have been painstakingly studied by Gye, Purdy, and Kettle Kettle has concluded from his experiments that silica, after it has entered the body, dissolves in a colloidal form in the weak alkalis of the lung fluid and is very gradually absorbed, the soluble portion then acting as a tissue poison. He thinks that through its toxic action it paralyzes the phagocytes so that they lose their phagocytic ability and also their power to deal with the tubercle bacillus, thus leaving the lung unprotected.[24] Kettle, Gye, Purdy, and S Lyle Cummins have all proved by experiment that far from being 'poisoned' by silica, the tubercle bacillus is stimulated by silica.[25]

Extensive studies have been made on the health of coal miners Dr Middleton, in a five year study of silicosis deaths reported in British industries, showed that during the period from June 1, 1931 to December 31, 1935, there were 147 certified deaths from silicosis and silicosis with tuberculosis and 511 total disablement cases among 139,228 employed coal miners in South Wales and Monmouth. In all the rest of England and Scotland, there were only 22 deaths and 70 total disablement certificates for silicosis in coal miners, in a total of over 600,000 employed.[26] Physicians practicing in the

South Wales coal fields believe the silicosis observed there is of the 'infective' type; 'infective' is Dr. Irvine's term for the type of extensive fibrosis found in silicotic lungs which is suspected of being produced by the aid of the tubercle bacillus, but in which the bacillus cannot be demonstrated.[14] Investigations in the iron mining industry disclosed a rather high silica content in hematite miners' lungs at autopsy and Stewart and Faulds felt justified in naming this lung condition 'sidero-silicosis.'

In 1933, W R Jones brought forward the theory that mineral sericite (a hydrous silicate of aluminum and potassium) was the chief factor in the development of silicosis.[28] However, this has not been substantiated Silicosis has been demonstrated in experimental animals and in human cases in the absence of sericite and where pure quartz was the only causative factor. It is interesting to note that many attempts have been made to find an 'antidote' dust to neutralize or delay the harmful action of silica Carleton,[29] in experiments with combined flint and coal dust on guinea pigs, found that coal dust appeared to cause enough initial immediate irritation to promote elimination of some, but not all, of the flint dust inhaled No definite conclusions have as yet been reached in this work which is still being conducted.

Compensation for workers disabled as a result of silicosis or silicosis and tuberculosis has been provided for in England by amendment to the compensation law. From 1925 to date, five schemes have been put into operation dealing with silicosis in various industries, including the refractories, metal grinding, sandstone, tin mining, coal mining, potteries, granite working, and others. Processes not involving exposure to silica dust are exempted, in some cases, the limits of the silica content being specified for the purpose of the law. The most important feature of these schemes is the provision for medical certification of physical fitness for work in such exposure,

for medical certification of disability with limited compensation for non-disabling stages of uncomplicated silicosis, and for post-mortem examination in death claim cases.

### Germany

In Germany, Paracelsus was first to deal with injury to the lungs of miners caused by inhalation of dust and to designate such injury as an occupational disease. In the nineteenth century, German pathologists and clinicians made considerable progress in regard to research connected with diseases caused by dust. Arnold succeeded in producing experimental dust fibrosis of the lung and in distinguishing clearly between that and tuberculosis Eulenberg definitely affirmed that silicic acid causes serious fibrotic disease of the lungs. The connection between inhalation of dust with quartz content and tuberculosis has been shown statistically by various writers: *Oldendorff, Sommerfeld, Moritz and Ropke* and others showed the high mortality rate from tuberculosis among metal grinders, Lewin, Hirt, Wilbrandt, and others drew attention to similar conditions among pottery workers.

Baumler described the symptoms of silicosis and stressed the cirrhotic character of the accompanying tuberculosis. Staub-Otiker described in detail the roentgen-ray appearance of the silicotic lung as found in metal grinders and also distinguished the various stages of the disease. *Stahelin, Ickert,* Jotten, Arnoldi, Lehmann-Engel-Wenzel have all given comprehensive expositions of this subject. Koelsch and Kaestle designated the appearance of fine mottling on roentgenogram as the first stage of silicosis, associated with slight dyspnea after effort. While it is admitted that very often tuberculosis is associated with dust fibrosis, the majority of German investigators are of the opinion that fibrosis due to dust is a disease in itself and to be distinguished from tuberculosis

and that dust inhalation, as such, may lead to extensive pulmonary fibrosis without associated tuberculous infection Schmorl, Staub-Otiker, Arai, Koopmann, Bohme, and Schridde describe such cases Merkel has concluded from his anatomical findings that simultaneous inhalation of dust and tubercle bacilli occurs and that these exert a double action in the production of the majority of dust fibrosis A great deal of experimental work has been done in Germany regarding silicosis by Lehmann, Gross, Jotten, and others

In 1929, 'severe pneumoconiosis' (silicosis) was included in the list of occupational diseases subject to compulsory compensation In the case of severe dust fibrosis combined with tuberculosis of the lungs, tuberculosis is regarded as pneumoconiosis (silicosis) for the purposes of compensation

Up to the beginning of the present century, all changes of the pulmonary tissue caused by different kinds of dust were considered as fairly similar However, the observations of the last few decades, particularly the thorough investigations effected in the last twenty years, have fundamentally altered the former theories Today it is recognized that the various types of dust must be judged in a different manner and that the decisive factor regarding effect is not the morphological physical activity of the various types but rather the content of free crystallized silicic acid (quartz) in the dust. Koelsch began some years ago to investigate conditions in dusty industries and in the last few years, over 1,200 workers in various branches of industry involving exposure to mineral dust were examined clinically and roentgenologically It was found that the most dangerous occupations in regard to pneumoconiosis, as related to dust exposure, were granite and sandstone working The statistical morbidity and mortality returns as well as the clinical roentgenological examinations,

show clearly the fundamental principle that the greater the quantity of free silicic acid in the dust, the more dangerous it is for the lungs [15]

## AUSTRALIA

The rapid increase in Australia's population in the latter half of the last century was due in large part to the discovery of rich alluvial gold fields in several parts of the Continent. This stimulated prospecting and brought about the discovery of other metalliferous deposits. A large portion of the migrants came from mining districts in England and other countries.

Silicosis has been demonstrated in many of the mining fields and a high death rate from pulmonary diseases, including tuberculosis, has been evidenced in connection with the industry.[15]

In 1902, while a system of sewers was being dug in Sydney, Australia, a Sewer Works Ventilation Board was appointed to inquire into the working conditions and to recommend means of improvement to render the work less hazardous. The board found that miners employed in this type of work *suffered from a disease known as 'sewer disease' but which* was strongly suspected of being due to dust. It was the board's opinion that although fumes from explosives, poor ventilation, etc, were contributory to the high mortality, the sole cause of the disease was probably the dust from hammering, drilling, and use of the pickax

The Australian government has made many investigations of the mining industry throughout the country and vigorous efforts have been made to control unhygienic conditions The chief metal mining sections in which health conditions have been surveyed are . Bendigo, Victoria, Broken Hill, New South Wales, Kalgoorlie, Kimberley, and Coolgardie in Western Australia.[14]

In 1912, Armstrong investigated a reported epidemic of pneumonia at Broken Hill and found the death rate from pneumonia among underground miners in that locality during 1910–2 was nearly 4 times as great as that for all males in New South Wales In 1914, the report of a Royal Commission investigating the mining industry at Broken Hill stated that although the prime cause of pneumonia was sudden changes in temperature, dust inhalation in any form would predispose to disease.[30]

In 1919, the Technical Commission constituted to examine the miners at Broken Hill used the roentgen-ray, for the first time in Australia, as a means of diagnosing silicosis as an occupational disease. The Commission made an elaborate report in 1921 and set up standards for diagnosing the different stages of silicosis [31]

In 1924, a very complete investigation of the sewer system situation was made by Dr Charles Badham, Industrial Medical Officer of the New South Wales Department of Health. Dust counts were taken and it was found possible to set a standard for permissible dust concentrations of 200 particles per cubic centimeter (approximately 5,664,000 particles per cubic foot)

A special scheme for the compensation of silicosis for Sydney sandstone workers was put through in 1927 and the whole field was checked again, surveys were also made among sandstone masons and quarriers in the neighborhood These were found to be exposed to much the same hazard as the tunnelers Open sheds and wet methods were recommended for controlling the dust hazard [14]

Since 1926, the mine employees in Western Australia have been examined annually by the Commonwealth Department of Health Of 2,290 men previously classed as normal, 1.3 per cent were found on re-examination in 1927 to be suffering from uncomplicated silicosis and 0 5 per cent had tubercu-

losis only, of 491 diagnosed as silicotic, 86 (17.5 per cent) had silicosis with tuberculosis. In 1929, of 2,293 normals re-examined, 100 were diagnosed as silicotic. *Silicosis was not* found in any case under forty years of age or with less than five years of underground work.[30]

## ITALY

Italian research workers have furnished interesting contributions to the knowledge of respiratory diseases resulting from the inhalation of dust. While experimental work was done by Biondi, Tommasi-Crudeli, Feliziana, and others, the report of Devoto and Cesa-Bianchi in 1911 must be considered especially notable. In their experiments with guinea pigs, they found that animals subjected to the inhalation of very fine limestone dust are less susceptible to tuberculosis than those which have inhaled silica dust.[15]

Among other workers who have studied the occurrence of pneumoconiosis in various Italian industries are Pesenti, Rota, and Finzi, who reported on health conditions of workers in lime, cement, and plaster; Frongia, who noted that the cause of death among Sardinian miners was most frequently an acute infection (pneumonia); Bianchi, who made careful clinical and radiological examination of workers employed as sculptors, hewers, modelers, and workers in grinding rooms. In regard to the importance of silica dusts as the cause of pneumoconiosis, a report by Giglioli is of real importance. Pieraccini had already remarked that among the workers in the Cornacchino mine, there occurred a high incidence of broncho-pulmonary affections and pulmonary tuberculosis; a later inquiry by Puccinelli and Ginanneschi confirmed this fact. Giglioli found that in many of the miners at Cornacchino, and in almost all who worked where the air is rich in silica dust, there occurred, after two to three years of work, broncho-pulmonary affections, with coughing,

breathlessness, emphysema, and in general, after a more or less lengthy period, all the signs of pulmonary tuberculosis. There is almost always a well-marked, pre-tuberculous period, which may evolve slowly without tuberculous complications corresponding to so called miner's phthisis. More recently, Mazzi, in 1913, on the basis of animal experiments and clinical observations, has come to the conclusion that silicon (in the special form of silicates) causes chronic intoxication of the system and acts especially on the blood in exercising a general anemia producing action.[15]

## Canada

In 1875, Sir William Osler, in one of his lectures, demonstrated two specimens of miners' dusted lungs. His description of the pathology of this disease compares favorably with modern descriptions of silicosis. Pneumoconiosis or silicosis or miner's phthisis has been scheduled as a compensable occupational disease in quarrying, stoneworking, metal grinding or polishing, and mining for a number of years in Ontario, Alberta, and Saskatchewan.

Silicosis has been studied from time to time in the gold fields of Ontario, at Porcupine, Kirkland Lake, Cobalt and Sudbury. Granite cutters in Ontario were examined in 1928. Other examinations have been made among foundry workers and among sandblasters, as well as workers in marble, talc cement, brick, grain elevators, and artificial abrasives. These investigations were carried out by the Industrial Hygiene Division of the Ontario Health Department, where Cunningham, Riddell, and their associates have done work on correlating clinical roentgen ray, and pathological findings in lungs of workers. Belt of Toronto University has made comparisons between the silica content of the ash of silicotic lungs and the number of particles to be seen in the same lungs with a polarized microscope. The method brings out 'invisible

silica' so often predicated by pathologists working on silicosis.

Outstanding experimental research on silicosis is being carried out at the Banting Institute of the University of Toronto, under the direction of Banting, by Doctors King, Fallon, Irwin, Stantial, Franks, and others. Irwin has developed a micro-incineration method which makes it possible to determine amounts of silica in tissues with considerably greater accuracy for the purpose of relating it quantitatively to the degree and location of fibrosis present in the lungs.[14]

Recent experimental work by Denny, Robson, and Irwin showed that animals dusted with quartz, to which less than 1 per cent of metallic aluminum dust had been added, showed practically no fibrosis.[32]

## UNITED STATES

As early as 1887, Dr. Frederick Peterson[14] of Poughkeepsie,[33] New York, presented a report of an autopsy upon a foundry worker which unmistakably indicated silicosis and called attention to the prevalence of this type of disease among fellow employees of his patient. In the same year, a series of patients employed in a cutlery factory were presented to the Franklin District Medical Society by Dr.F.J Canedy of Shelburne Falls,[14] Massachusetts.[34] Dr.William W.Betts of Nevada, in 1899,[14] described[35] a series of cases of pulmonary disease among employees of a gold crushing plant.

Not until 1915 was the first study of silicosis and allied disorders initiated by the United States Bureau of Mines and the United States Public Health Service, jointly, in the lead and zinc mining district of southwest Missouri.[36] This investigation was followed by similar studies in hard rock metal mining regions in other parts of the United States[37]

In 1924, the United States Bureau of Mines established a clinic in the Tri-State Lead and Zinc mining district (Missouri, Kansas, and Oklahoma) which was later (1927) ex-

panded into a co-operative enterprise by the Bureau of Mines with the Tri-State Producers Association and the Metropolitan Life Insurance Company This clinic was maintained for nine years Over 60,000 complete physical examinations of 27,000 individuals were performed at this clinic while it was maintained under Government auspices, including roentgenogram of the chest and a blood Wassermann or Kline test The results are partially summarized in Technical Papers 545 and 552 of the United States Bureau of Mines.[38] The general tenor of these studies in the United States was in close conformity with those of South Africa and England

Attention was later directed to the anthracite coal mining industry, in which a disabling pulmonary disease, locally known as miner's asthma, had long been recognized In 1933, the United States Public Health Service, in co-operation with the coal mine operators, the United Mine Workers of America, and the Pennsylvania Department of Labor and Industry, undertook a survey of pulmonary disease in the anthracite mines, the results of which were published in Bulletin 221 of the United States Public Health Service [39] This study confirmed what many had suspected — that disabling pulmonary fibrosis among coal miners was due to silica and not to coal dust, although the coal might modify the clinical and pathological picture

Other studies of the United States Public Health Service in bituminous coal mines failed to establish a disabling fibrosis in the absence of silica Brundage of the Public Health Service had demonstrated from mortality records that bituminous coal miners had a low rate of tuberculosis To the condition found in the anthracite mines where silica bearing rock is often adjacent to the coal Sayers and his colleagues gave the name anthraco-silicosis and demonstrated that in the older age groups, the incidence of tuberculosis as a complication was very prevalent In the age group forty five to fifty-five,

the tuberculosis rate was five times that of the general population and above age fifty-five, ten times as large.[39]

About the same time, Gardner and Cummings of the Saranac Laboratory started their extensive studies of pulmonary disease in the iron ranges of northern Michigan and Wisconsin. The general conclusions of all these field studies indicated that the incidence of silicosis and the resulting clinical picture were directly related to the severity of exposure to quartz dust and to the presence of infection and that other substances, in combination with the silica, might and often did modify the action of the silica.

These conclusions are supported by the results of investigations of foundries. Foundry studies were conducted at various times by the United States Public Health Service,[40] by the Industrial Hygiene Division of the Metropolitan Life Insurance Company,[41] by the Saranac Laboratory and the American Foundrymen's Association,[42] the State of Connecticut,[43] and the State of Massachusetts.[44]

A number of investigations were also made in the granite industry, notably in 1918 by the Committee on Mortality from Tuberculosis in the Dusty Trades,[45] under the leadership of Dr Frederick L Hoffman, and later, in 1929, the important study by Russell of the Public Health Service.[46]

The sandstone industry was investigated by Hayhurst in 1926,[47] the marble industry by Dreessen of the Public Health Service in 1934;[48] and the pottery industry on a number of occasions[49]

The cement industry also has been thoroughly studied, beginning in 1915 by Tucker,[50] and later by the Public Health Service[51] and also by the Saranac Laboratory[14] These various investigations of the cement industry revealed little or nothing in the way of occupational pulmonary disease and served to confirm the so-far accepted principle that disabling

pulmonary fibrosis is dependent upon exposure to the du[st] of uncombined or 'free' silica and not 'silicates.'

The manufacture of abrasives received considerable atten[tion] as did metal grinding The United States Public Health Service,[52] the United States Department of Labor, Dr. C.-E.A. Winslow [53] in Connecticut, Dr W Irving Clark [54] in Massachusetts, Dr. H H Kessler [55] in New Jersey, and others have reported on the abrasives industry, and Winslow and Greenburg,[56] W H Drury [57] and others on metal grinding

During the years that these investigations were carried on, much laboratory and clinical research was under way. In Philadelphia, Stengel, Pancoast, Landis, T Grier Miller, and Henry Field Smyth, interested in tuberculosis in industry, and conducted studies in the relationship of industrial dusts pulmonary disease, numerous articles and reports came from this group, both collectively and individually, commencing about 1914

During the years 1914–30, in association with the Bureau of Mines and later on their own behalf, the Public Health Service combined field studies with laboratory research in an effective manner Miller in the Public Health Service demonstrated the technique of intraperitoneal injections of silica and other dusts to determine the tissue reaction [59] Gardner and his associates at the Saranac laboratory demonstrated that attenuated tubercle bacilli in the presence of quartz dust would, in experimental animals, produce fatal tuberculosis without increasing the virulence of the bacilli [58]

In 1922, Greenburg and Smith introduced the impinger, a dust sampling device, which resulted in the impinger method becoming the standard for the United States Bureau of Mines and the Public Health Service [60]

The intimate relationship of roentgenological diagnosis to the silicosis problem was recognized early In 1926 Pancoast

and Pendergrass published the first book to appear on this subject[61] These authors, together with Sampson and others, have carried on experimental work in technique and in elaborating standards of diagnosis.[62]

The determination of disability in silicosis has proved vexatious to both clinicians and workmen's compensation officials. At the University of Rochester, McCann and his associates have been working on physiologic methods of estimating pulmonary impairment.

Harrington of the United States Bureau of Mines, Philip Drinker at the Harvard School of Public Health, Cummings at Saranac, Bloomfield and Dallavalle of the Public Health Service, Fehnel and his associates at the Metropolitan Life Insurance Company, have contributed a very large amount of work to the engineering and chemical methods of dust determination and control Here again, the reports and publications are far too numerous to detail and the reader is referred to the original sources.

In addition to all the publications made available by all this field and laboratory work, the Harvard School of Public Health, the Saranac Laboratory, and the Division of Industrial Hygiene of the United States Public Health Service have undertaken direct educational activities A recent organization is the Air Hygiene Foundation, sponsored by various industries to encourage research into atmospheric pollution and pulmonary disease and to carry on educational work

## 2 HISTORY OF ASBESTOSIS

Asbestos was mined and the fibres were separated from the mineral and used in the making of fire-proof fabrics several centuries B C Attention is called by Cooke[63] to a cremation cloth described by Herodotus in 450 B C. Materials made of asbestos have been used ever since but in the past three decades, the industry has grown tremendously owing to the

greatly increased use of fire resisting and insulating materials as well as brake linings in the automobile industry

Asbestos is a magnesium silicate, containing a small amount of iron and a trace of aluminum. The fibres are long and flexible and for many purposes are combined with cotton to undergo regular textile processes of spinning and weaving Most of the asbestos used in the United States comes from Canada.

The first report of a death resulting from asbestos dust inhalation was presented by Murray in 1900[64] A second case was reported by Cooke[65] in 1924 and again in 1927.[63] At that time, Cooke described the unusual and characteristic asbestosis bodies In 1927, Oliver[66] reported 2 cases, to be followed by McDonald[67] and Seiler.[68] In 1928, Simson[69] reported 2 cases in South Africa Other cases were reported by Wood and Page[70] in 1929, Haddow[71] in 1929, and Wood and Gloyne[72] in 1930, in that year appeared the comprehensive report of Merewether and Price[73]

In the United States, Lynch and Smith[74] reported the first cases in 1930 The first investigation of the asbestos industry in the United States was made in 1930 and 1931 by Lanza, McConnell, and Fehnel.[75] In the last few years, much attention has been given both to the clinical aspects and diagnosis of asbestosis and to its pathology In 1936, Shull reported a roentgenological review of 71 cases.[76] Recently the United States Public Health Service[77] has published several reports of dust control methods in asbestos plants and of the industry in general

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## II. ETIOLOGY, SYMPTOMS, DIAGNOSIS OF SILICOSIS AND ASBESTOSIS

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### 1 ETIOLOGY, SYMPTOMS, AND DIAGNOSIS OF SILICOSIS

SILICOSIS has been variously defined Dorland ( Dict 16th ed pp 68 ) 'Pneumoconiosis due to inhalation of the dust of stone, sand, or flint ;' ( Webster's New International Dictionary, 1930, p 1956 ) 'An affection of the lungs occurring in stone cutters, caused by the inhalation of quartz dust '

The Committee on Pneumoconiosis of the Industrial Hygiene Section of the American Public Health Association, recently defined silicosis as 'a disease due to breathing air containing silica ( $SiO_2$ ) . characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis ( some or all of which symptoms may be present ) , and by characteristic X-ray findings ' [1]

The compensation law of West Virginia defined silicosis as 'an insidious fibrotic disease of the lung or lungs, due to prolonged inhalation and accumulation sustained in the course of and resulting from employment, of minute particles

of dust containing silicon dioxide ($SiO_2$) over such a period of time and in such amounts as result in the substitution of fibrous tissue for normal lung tissues; and the term silicosis shall also include silicosis accompanied by tuberculosis of the lungs evidenced by the presence of tubercle bacillus in the sputum '[2]

While the foregoing definitions express somewhat different viewpoints, they have this in common — the cause of silicosis is silica or quartz. Although other dusts, when inhaled in sufficient concentrations over a long enough period of time, have been shown capable of producing a definite pulmonary fibrosis, nevertheless the pneumoconiosis characterized by nodular fibrosis has to date been shown clinically and experimentally to be associated only with the inhalation of dusts containing free or uncombined silica.

## Silica in Nature

Silica is the most abundant constituent of the minerals and rocks that make up the crust of the earth. It occurs in two forms, free and combined The free silicas, as a group, are definite compounds in the form of $SiO_2$ The combined forms are silicates. Of free silicas which occur in nature, that known as quartz is by far the most common Quartz is a hard mineral and chemically resistant to reagents. It is an abundant constituent of granite, schist, and other rocks, and the chief component of sandstone and quartzite Many ores are deposited in veins that consist nearly wholly of quartz. Probably the next most common form in which free silica exists in nature is the amorphous hydrated form known as Opal ($SiO_2 \cdot H_2O$). Opal is a silica of colloidal origin and occurs abundantly in the diatomaceous earths It is less resistant to reagents than quartz Another type of free silica is flint and with flint is found chalcedony, a waxy, translucent form of silica interpreted as consisting of fibres of quartz with a small

amount of interstitial opal. Other forms of free silica occurring less abundantly in nature are tridymite, cristobalite, and siliceous glass or vitreous silica

### Occupational Exposure to Silica

As the earth's crust contains so large an amount of silica, it is obvious that those occupations concerned with mining and the driving of tunnels are associated with a silicosis hazard Other occupations exposing the workers to this hazard are those concerned with the processing and industrial use of mineral products, such as smelting and refining, the use of sand and gravel for structural purposes, the cutting of stone, particularly granite, the manufacture and use of certain abrasives; and the processing of the various forms of free silica According to Knopf,[3] the most common forms of free silica used industrially are massive crystalline quartz, quartzite, sandstone, flint, tripoli, diatomaceous earths and silica sand. Table 1, from Ladoo,[4] illustrates the great variety of uses to which silica is put in industry and the kind of silica adapted to each purpose

TABLE I

| USES OF SILICA | TYPES OF SILICA USED |
|---|---|
| Abrasive uses | |
| In scouring and polishing soaps and powders | Quartz quartzite flint, chert sandstone, sand, tripoli, and diatomaceous earth, all in finely ground state |
| In sandpaper | Quartz, quartzite flint sandstone and sand coarsely ground and closely sized |
| In sand blast work | Quartz quartzite sandstone and sand crushed into sharp angular grains uniform in size |
| Metal buffing, burnishing and polishing | Ground tripoli and other forms of ground silica. |

| USES OF SILICA | TYPES OF SILICA USED |
|---|---|
| For sawing and polishing marble, granite, etc | Sharp, clean sand graded into various sizes |
| As whetstones, grindstones, buhrstones, pulpstones, oilstones, etc | Massive sandstone from very fine to moderately coarse grained |
| Tube-mill lining | Chert, flint, and quartzite in dense solid blocks |
| Lithographers' graining sand | Medium to fine sand or rather coarsely ground silica and tripoli. |
| Tube-mill grinding pebbles | Rounded flint pebbles |
| In tooth powders and pastes | Various forms of pure silica finely ground. |
| Wood polishing and finishing | All forms of silica ground to medium fineness |
| Refractory uses | |
| In making silica fire brick and other refractories | Fairly pure quartzite known as ganister; not less than 97 per cent $SiO_2$ nor more than 0.40 per cent alkalis, tightly interlocking grains desired. |
| Metallurgical uses | |
| In making silicon, ferrosilicon, and silicon alloys of other metals, such as copper | Moderately pure sand, massive crystalline quartz, sandstone, quartzite, or chert |
| As a flux in smelting basic ores | Massive quartz and quartzite. |
| Foundry-mold wash | Ground sandstone, quartz, and tripoli. |
| Foundry parting sand | Fine sand and ground tripoli |
| Chemical industries | |
| As a lining for acid towers | Massive quartz or quartzite |
| *As a filtering medium* | Massive diatomaceous earth and tripoli, sand, finely granular quartz or quartzite, finely ground tripoli, diatomaceous earth, and other forms of silica |
| *In the manufacture of sodium silicate* | Pure pulverized quartz sand, pure tripoli, and diatomaceous earth |
| In the manufacture of carborundum | Pure quartz sand |
| *Paint* | |
| *As an inert extender* | Finely ground crystalline quartz, quartzite, and flint; also finely ground sandstone, sand, and tripoli. |

| USES OF SILICA | TYPES OF SILICA USED |
| --- | --- |
| Mineral fillers<br>As a wood filler<br>In fertilizers<br>In insecticides | Finely ground crystalline quartz, quartzite, flint, tripoli, and other types of ground silica |
| As a filler in rubber hard rubber pressed and molded goods, phonograph records, etc<br>In road asphalt surfacing mixtures | Finely ground silica of all types |
| Ceramic uses<br>In the pottery industry as an ingredient of bodies and glazes<br>In the manufacture of ordinary glass | Flint, tripoli, and chert and other amorphous silica preferred, also all other forms of very pure silica, all finely ground |
| In the manufacture of fused quartz chemical apparatus such as tubes crucibles and dishes | Very pure massive quartz preferred |
| Decorative materials<br>In the manufacture of gems, crystal balls, table tops vases statues etc | Rock crystal amethyst, rose quartz, citrine quartz smoky quartz chrysoprase, agate chalcedony opal, onyx sardonyx jasper etc |
| Insulation<br>Heat insulation for pipes, boilers, furnaces, kilns, etc<br>Sound insulation in walls, between floors etc. | Massive and ground diatomaceous earth |
| Structural materials<br>Sand lime brick | Moderately pure, sharp angular sand, preferably finer than 20 mesh, together with a small percentage of finely pulverized silica |
| Optical quartz<br>For the manufacture of lenses and accessories for optical apparatus | Clear, colorless, flawless rock crystal or massive crystallized quartz |

In a recent survey,[5] carried on in a large manufacturing center, it was found that about 9 per cent of the industrial workers were employed in occupations where the silica hazard required consideration According to the census from 1930, there were gainfully employed in the manufacturing and

mechanical industries in this country approximately 14,000,000 persons If the above survey can be accepted as representative of the occupational distribution of these workers, it would appear that there are nearly 1,200,000 individuals possibly exposed to a silicosis hazard in the manufacturing and mechanical industries alone Lanza and Vane,[6] in their discussion concerning the prevalence and effect of silicosis, state · 'Our very rough, but obviously conservative estimate of the number of workers exposed to silica dust to a harmful degree in the United States is, therefore, upwards of 500,000'

## Factors Influencing the Action of Silica Dust Particles as the Exciting Cause of Silicosis

Although it has been accepted that silica is the exciting cause of silicosis, there are certain factors which must be considered as influencing its action. Early workers were inclined to consider that the injury produced by the dust particle was due to the mechanical irritation of its hard and cutting edges Gardner[7] has shown experimentally that the inhalation of finely divided carborundum dust, which has sharp edges and a greater hardness than silica, does not produce the nodular reaction characteristic of silicosis Collis[8] was one of the early workers to draw attention to the chemical action of dust Gye and Kettle[9] have shown that silica in solution or non-crystalline form exerts a toxic action upon the tissues which leads to the proliferation of fibroblastic cells Lately, Miller and Sayers[10] have reported results of experimental studies and have classified the reaction of peritoneal tissues to certain dusts Only the silica containing dusts have uniformly produced the proliferative harmful reaction Other dusts have been either completely absorbed, leaving no scar tissue, or have remained unaltered in the form in which they were injected These latter reactions are classed as absorptive and inert

Since dust, to exert its harmful action, must enter the finer divisions of the lung, its particle size bears a definite relationship to its injurious effects The silica must be present in the air in particles small enough to enter the finer air spaces and of such dimensions that the phagocytic cells may engulf them. The natural defenses of the respiratory tract probably prevent many particles larger than 10 microns from reaching the finer divisions of the lung and such as do are likely to be expelled with the bronchial secretions. The solubility of the silica may play a definite part in the production of disease and the size of the particle also affects the rate of solution as the smaller the particles, the greater the total surface area exposed to the action of solvents.

Table 2 shows the size distribution of various industrial dusts as compared with the dust particles observed in the out door air in the general atmosphere About 70 per cent of the particles found in industrial dusts generally are between 0 5 and 3 microns in diameter There are, no doubt, a great many particles too small to count by the method used but experimentally it has been shown that such sub-microscopic particles are not retained in the lungs but pass out with the expired air Sayers [11] has shown that less than 15 per cent is retained when finer particulate matter, such as lead in the form of fumes, is inhaled. The majority of particles found upon microscopic examination of lung tissue also fall within the limits of from 1 to 3 microns

Another reason for considering the size of the particles as affecting the harmfulness of the dust, is that the larger ones settle out with comparative rapidity from the atmosphere while the rate of falling for the smaller particles is very slow Figure 1 illustrates graphically this difference , particles under 1 micron falling at the rate of from 1 to 3 feet per hour, varying with the specific gravity, while particles 5 microns in diameter and of specific gravity 7 fall about 60 feet per hour

# TABLE 2

## Size-Frequency Distribution of Various Industrial Dusts as Compared to Outdoor Dust

[ *Average frequency in per cent* ]

| Kind of dust | Number of Samples | Median | Size Group in Microns | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 0–0.49 | 0.5–0.99 | 1–1.49 | 1.5–1.99 | 2–2.49 | 2.5–2.99 | 3–3.49 | 3.5–3.99 | 4–4.49 | 4.5–4.99 | 5–5.49 | 5.5–5.99 |
| Outdoor dust | 179 | 0.5 | 56.0 | 41.0 | 2.5 | 0.5 | — | — | — | — | — | — | — | — |
| Sandblasting | 9 | 1.4 | 1.4 | 19.7 | 34.7 | 20.3 | 12.6 | 5.2 | 2.8 | 1.6 | 1.1 | 0.2 | 0.2 | 0.2 |
| Granite cutting | 4 | 1.4 | 2.0 | 19.0 | 33.6 | 24.5 | 10.4 | 4.6 | 3.1 | .6 | .9 | .3 | 1.0 | — |
| Trap-rock milling | | | | | | | | | | | | | | |
| Crusher house | 1 | 1.4 | — | 13.0 | 39.0 | 33.0 | 10.5 | 2.5 | 2.0 | — | — | — | — | — |
| Screen house | 1 | 1.3 | 2.0 | 31.5 | 33.0 | 16.0 | 10.0 | 4.5 | 2.5 | .5 | — | — | — | — |
| *Disc crusher* | 1 | .9 | 10.0 | 48.0 | 31.0 | 6.0 | 3.0 | 1.0 | 1.0 | — | — | — | — | — |
| Foundry parting *compound* | 2 | 1.4 | .5 | 22.0 | 42.0 | 17.3 | 9.2 | 5.0 | 1.5 | 2.0 | .5 | — | — | — |
| General foundry air | 1 | 1.2 | — | 26.0 | 48.0 | 17.0 | 8.0 | 1.0 | — | — | — | — | — | — |
| Talc milling | 1 | 1.5 | — | 16.0 | 32.0 | 20.0 | 13.0 | 7.0 | 5.0 | 2.0 | 2.0 | 2.0 | — | 1.0 |
| Slate milling | 1 | 1.7 | 1.0 | 13.0 | 29.0 | 17.0 | 14.0 | 14.0 | 6.0 | 4.0 | 1.0 | — | 1.0 | — |
| Marble cutting | 1 | 1.5 | — | 12.0 | 37.0 | 21.0 | 10.0 | 11.0 | 3.0 | — | 1.0 | 2.0 | 2.0 | 1.0 |
| Soapstone | 2 | 2.4 | 1.2 | 16.0 | 19.0 | 13.0 | 11.0 | 6.0 | 6.5 | 4.5 | 5.5 | 3.3 | 2.5 | 11.5 |
| Aluminum dust | 1 | 2.2 | 3.0 | 8.0 | 20.5 | 14.0 | 11.5 | 9.0 | 6.5 | 3.0 | 3.5 | 4.0 | 7.0 | 10.0 |
| Bronze dust | 1 | 1.5 | 1.0 | 12.0 | 33.5 | 25.0 | 21.0 | 6.0 | 1.5 | — | — | — | — | — |
| Anthracite-coal mining: | | | | | | | | | | | | | | |
| Breaker air | 2 | 1.0 | 7.0 | 51.0 | 26.0 | 8.0 | 3.0 | 3.0 | 2.0 | — | — | — | — | — |
| *Mine air* | 1 | .9 | 11.0 | 60.0 | 17.0 | 7.0 | 3.0 | 1.0 | 1.0 | — | — | — | — | — |
| Coal drilling | 1 | 1.0 | 1.0 | 55.0 | 34.5 | 7.5 | 1.5 | — | .5 | — | — | — | — | — |
| Coal loading | 3 | .8 | 11.5 | 56.3 | 24.3 | 5.5 | 1.5 | .7 | .2 | — | — | — | — | — |
| Rock drilling | 1 | 1.0 | 4.0 | 49.0 | 25.5 | 12.5 | 5.5 | 1.5 | .5 | 1.5 | — | — | — | — |

Particles of more than 10 microns settle out in a relatively short time.

Thus we may say that the harmfulness of a given dust is directly influenced by the number of particles it contains of free silica less than 10 microns in diameter and that probably the most damage is produced by those between 1 and 3 microns.

The relationship of dust concentration and duration of exposure is closely associated in their etiological significance. The rate at which silicosis will develop, excluding certain factors considered as predisposing, depends upon the dosage

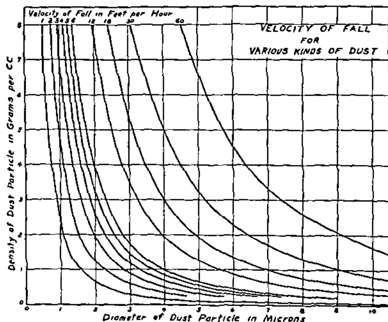



FIGURE 1

Diameter of Dust Particle in Microns — Density of Dust Particle in Grams per CC.

of free silica. This dosage is obviously dependent upon the amount of silica in the air inhaled and the duration of exposure. In this lies a point of great practical importance which explains the apparent discrepancies between the reports of different investigations with respect to the time necessary for silicosis to develop

In 1902, a committee, of which Dr.J.B S.Haldane [12] was a member, reported upon an investigation made to determine the cause of excessive mortality rates from tuberculosis among Cornish tin miners These investigators pointed out that it was evident that the inhalation of stone dust by these miners was the cause of permanent damage to the lungs. Furthermore, they noted that the condition developed gradually in the case of the ordinary miner but rapidly in the case of the machine workers who were exposed to greater amounts of dust. Dr.Watkins-Pitchford [13] and Dr.Mavrogordato,[14] as well as other South African investigators, emphasize the relationship of the concentration of dust and duration of exposure to the degree of lung changes produced. Intermittent employment has been suggested as an aid in preventing silicosis but aside from the fact that it does delay the ultimate result, it has not been shown that such interruption of work can safely be relied upon where harmful concentrations of dust are involved In the United States, the reports of the Picher Clinic [15] have indicated the effects of varying periods of exposure to silica in the mines of that district. Similar studies of the health of granite workers [16] [17] likewise stress the duration of exposure necessary to produce definite degrees of silicosis

Various investigators have stated that the presence of other substances in silica containing dust may influence the action of silica tending either to aggravate or retard the usual reaction. The question was raised by Heffernan [18] in 1926 as a result of a study of a group of brick makers in Derbyshire He

concluded that the clay present in the silica dust was responsible for the absence of silicosis in this group of workers. Gardner is strongly of the opinion that the action of silica is modified by other substances which may be in combination with it and this is supported by the recent Canadian reports with reference to the action of metallic aluminum [19] What the implication of this may be with respect to specific dust problems in various industries remains to be determined.

Cases of so-called acute silicosis, resulting from the presence in highly siliceous dust of alkali in a fine powdered form, have been reported by Chapman,[20] Kessler,[21] MacDonald,[22] Kilgore,[23] and others Some have suggested that the action of silica is made more rapid by the presence of these alkalis because silica is more soluble in alkaline solutions. Kettle [24] failed to demonstrate such action experimentally and states that proof is still lacking that any action of the kind may occur The effects of inhaled coal dust in conjunction with silica have been reported at length by the Public Health Service [25] in their study of anthraco silicosis among hard coal miners

Taken all in all, the available data indicate that other components of silica laden dust are not merely inert diluents whose effect is to reduce the amount of silica inhaled Some of these substances may act in this manner others apparently aid in removing the silica particles from the atmosphere, and others may accelerate or retard the action of silica. Moreover, when inhaled with silica, these latter tend to alter the anatomical form of the silicotic lesion The degree of modification is determined by the character and amount of the adulterant dust

## Occupational History

Most important in determining the significance of a given dust exposure is a complete, carefully taken occupational

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## Occupational History

Most important in determining the significance of a given dust exposure is a complete, carefully taken occupational

history, whereby the total dust exposure may be estimated and compared with other cases of a similar nature. Tables 3, 4, and 5 illustrate a practical method of analyzing and recording occupational factors in the study of a dusty industry.[25]

TABLE 3

OCCUPATIONAL RECORD

Division of Industrial Hygiene — United States Public Health Service

Dated August 8, 1933 — Office Industrial Hygiene
Name K M — Present age 49
Age began work 15 — Number of years worked : 34

| | Specific Occupation | Specific Industry | No of years in Hard Coal | Non dusty |
|---|---|---|---|---|
| Present | Section foreman | Anthracite coal | 5 | |
| Preceding | 1 — Contract miner (chamber) | " " | 15 | |
| | 2 — Miners' laborer (chamber) | " " | 3 | |
| | 3 — Mule driver (dry mine) | " " | 3 | |
| | 4 — Patcher (dry mine) | " " | 2 | |
| | 5 — Slate picker (dry breaker) | " " | 2 | |
| | 6 — Farm laborer (Pa) | Agriculture | | 1 |

Remarks Estimating the total time idle during working life — 3 years

Table 4 shows the occupations and associated dust exposure of chamber miners and laborers. Each occupation engaged in by these coal miners was studied separately. Experience has shown that the various occupations comprising the activity of any dusty industry are usually associated with dissimilar dust exposure.

Table 5 shows a method of analysis used in estimating an individual's total dust exposure

With such occupational histories available and the knowledge of the percentage of total dust exposure revealed by detailed occupational analysis, it was found possible in a recent study to predict in 9 cases out of 10 approximately the

## TABLE 4

### Dust Exposure of Contract Miners and Helpers (Laborers)

### Chamber Mining

| *Occupation* | *Number of Samples* | *Number of Hours in Activity* | *Average Dust Count (millions of particles per cubic foot of air)* | *Millions of Particle-Hours Per Cubic Foot* |
|---|---|---|---|---|
| Jack-hammer drilling | 23 | 1 | 575 | 575 |
| Hand loading | 22 | 2 | 1,138 | 2,276 |
| After firing | 7 | ¼ | 834 | 209 |
| Taping and wiring | 2 | ½ | 40 | 20 |
| Setting up props, and in main airways | 8 | 2¾ | 15 | 41 |
| Totals | 62 | 6½ | — | 3,121 |

$\frac{3{,}121 \text{ million particle-hours per cu ft}}{6\frac{1}{2} \text{ hrs}}$ = 480 million particle-hours per cu ft.

pulmonary condition that would be found upon physical examination, insofar as changes due to dust inhalation were concerned.

A statement on the effect of variations and concentrations and composition was developed by the Committee on the Prevention of Silicosis through Medical Control.[26] An abstract of this report follows

The effects of the variation in concentration of silica in the air breathed must be judged by data from those field and laboratory studies which have reported results that can be compared The following concentrations have been accepted as permissible for particular industries in the localities indicated

'It is believed that the effects of other substances associated

with silica in atmospheric dusts are sufficiently marked and well defined to preclude comparison of silica hazards merely on the basis of the amount of free silica in the atmosphere. It is concluded that atmospheric concentration of silica, which may have proved incapable of producing silicosis in one industry, will not necessarily be harmless under other conditions. The reverse is equally true. If these premises are as

### TABLE 5

ESTIMATION OF THE INDIVIDUAL'S TOTAL DUST EXPOSURE

Name K M

| *Occupation Foreman* | *Number of Years* | *Dust Concentration in Millions of Particles Per Cubic Foot (average)* | *Millions of Particle-Years Per Cubic Foot* |
|---|---|---|---|
| Slate Picker (dry mine) | 2 | 380 | 760 |
| Patcher (dry mine) | 2 | 71 | 142 |
| Mule driver (dry mine) | 3 | 71 | 213 |
| Miner's laborer (chamber) | 3 | 480 | 1,440 |
| Contract miner (chamber) | 15 | 480 | 7,200 |
| Section foreman | 5 | 7 | 35 |
| Total | 30 | — | 9,790 |

$\frac{9{,}790 \text{ millions of particle-years per cu ft}}{30}$ = 326 millions of particle years per cu ft

sound as they seem, there can be no universal regulatory standard of permissible dust concentration at the present time. Separate standards must be set for different industrial dusts and processes When all of the factors determining such standards have been more accurately measured and correlated it may become possible to use them as a basis for the general regulatory standard

'It is recommended that clinical and engineering studies be carried out as rapidly as possible to establish the necessary

scientific data upon which we may base practical and effective regulatory standards

'The factors necessary to establish a safe concentration of atmospheric dust are the following

'1. The relative number and size of the free silica and the other particles suspended at breathing levels in the different parts of the industrial atmosphere

| *Industry* | *Percentage of Silica in the Dust* | *Permissible Maximum Safe Dust Concentration Millions per cubic foot* |
|---|---|---|
| (1) South Africa Gold Mines | 80% | 4½ |
| (1) Ontario Gold Mines | about 35% (in the rock) | 8½ |
| (1) Australia Sandstone | 90% (in the rock) | 6 |
| (2) Barre Granite | 31% to 35% | 10 to 20 |
| (2) Pennsylvania Anthracite Mines | 35%<br>13%<br>5% | 5 to 10<br>10 to 15<br>50 |
| (1) Broken Hill, Australia Lead-Zinc Mines | 10% to 17% | 14 |

(1) Based on engineering practice (2) Based on clinical studies

'2 The proportion of the workmen's time actually spent n the various dust concentrations

'3 The effects of exposure to environment, as revealed by ne occupational and medical histories of old employees, by hysical examination and by the roentgenological appearance their lungs

From the viewpoint of silicosis prevention, the standard safe atmospheric dust concentration is the maximum in ch persons can spend their entire industrial lives without

producing demonstrable evidence of significant reaction in their lungs.

'There is evidence that for prolonged exposure, a concentration of more than 5,000,000 particles per cubic foot of a highly siliceous dust is dangerous Therefore, it is now considered good practice to hold concentrations of highly siliceous dust at 5,000,000 particles per cubic foot or less

'Since standards of safe atmospheric dust concentration, based on medical findings, have been established but for a few industrial dusts, and in view of the fact that to establish such standards for other industrial dusts will require considerable study and investigation, some arbitrary standard to serve tentatively as a measure of good practice may be useful. This arbitrary standard should be based upon what is believed to be within the limits of good engineering practice and that which, from a medical viewpoint, it is judged will largely control the silicosis hazard for most industrial exposures It has been suggested that the maximum permissible concentration of silica in the air breathed might be expressed by the following formula (Determination of dust concentration according to the technique described by the United States Public Health Service in Reprint 1520 from Public Health Reports, March 18, 1932). Multiply the percentage of free silica by the total particle dust count. If the result is under 5,000,000, the condition may be considered permissible. If the result is over 5,000,000, the condition may be considered too high For example, 10 per cent free silica with an average total dust concentration of 30,000,000 particles per cubic foot would give 0 10 times 30,000,000 which equals 3,000,000 (good practice); 30 per cent with an average total dust concentration of 50,000,000, would equal 0 3 times 50,000,000 or 15,000,000 (unsatisfactory). This formula is not applicable to any dust containing less than 3 per cent free silica.'

## Predisposing Causes

The wide distribution of silicosis indicates that all races are susceptible and that no nationality is exempt Although available data show that the incidence of silicosis is higher among the younger miners in districts where the percentage of free silica is high and among older miners where the percentage of silica is low, age, in itself, is probably not a factor The history of previous dust exposure and of previous respiratory infection is obviously of the utmost importance in the study of either an isolated case or of an industrial group Individual susceptibility is frequently mentioned but the weight of evidence is that it may be considered an acquired and not a congenital condition

Lehmann's [27] experiments to determine the functional efficiency of the upper respiratory tract, in the removal of dust, suggest that abnormalities of the nasal passageways may affect the time element in the development of silicosis

Infections of the upper respiratory tract may be of importance Sinus infections may act by decreasing the efficiency of the upper respiratory tract in the removal of dust from the air passing to the lungs and also may be responsible for the spread of infection to the lower respiratory tract Acute pneumonic conditions as well as the more chronic lung changes such as chronic bronchitis, bronchiectasis, and bronchiolectasis, emphysema, and pleurisy, all tend to lessen the ability of the lung to rid itself of foreign materials, through impaired lymphatic drainage and inability to force the bronchial secretions and foreign matter from the lungs

## Symptoms

The Committee on the Prevention of Silicosis through Medical Control [26] has summarized the symptoms associated with silicosis both in the presence and in the absence of infection, as follows (condensed) ·

## Subjective Symptoms

*Dyspnea.* The complaint most frequently mentioned is shortness of breath. Depending upon the extent of the involvement, this varies from slight dyspnea, following exertion, to marked dyspnea upon the least exertion or even when at rest The shortness of breath noted in silicotics presents one peculiarity in that it is seldom accompanied by orthopnea, the individual being no more short of breath lying down than when in an upright position This may not be so, however, when silicosis is complicated by cardiac disease or by true asthma

| *Silicosis* | *Silicosis with Infection* |
| --- | --- |
| Noted as a rule only after sudden or extra exertion Seldom so marked as to interfere with routine duties However, in cases with extensive pulmonary fibrosis, it may limit the individual's activities | If complicating infection is not widespread, may be no more marked than in cases of simple silicosis but as infection and fibrosis increase, it becomes disabling. |

*Cough* Many silicotics complain of a troublesome cough This cough differs from that resulting from simple irritation due to dust which clears up upon removal from exposure When present, it is more pronounced in the morning or upon beginning work after a rest period.

| *Silicosis* | *Silicosis with Infection* |
| --- | --- |
| The typical silicotic cough is dry and non-productive It usually parallels shortness of breath in degree and may contribute to disability | The cough usually becomes more troublesome and is productive The sputum varies from thick, tenacious, mucous material to that of a foul, purulent or purohemorrhagic consistency |

| | |
|---|---|
| | Microscopic examination or animal inoculation frequently reveal tubercle bacilli or, in some cases, organisms of the fusiform spirochetal group In advanced cases, coughing attacks are often of such severity as to leave the individual exhausted. |

*Chest Pain* This symptom is complained of by a majority of silicotics It varies from a feeling of tightness in the chest to the sharp pain typical of pleurisy (Since chest pain is offered as a complaint in many conditions, it cannot be stressed as especially characteristic of silicosis )

| *Silicosis* | *Silicosis with Infection* |
|---|---|
| A late symptom in cases of simple silicosis Then seldom more than a sense of tightness or feeling of substernal pressure. | Pleuritic pain is suggestive of a complicating infection It is increased by exercise and by coughing and may be distressing in advanced cases with extensive infection |

*Hemoptysis.* True hemoptysis seldom occurs Frequently, however, the sputum may be blood-streaked following a severe coughing attack Hemoptysis must always be considered as suggestive of tuberculosis

| *Silicosis* | *Silicosis with Infection* |
|---|---|
| Occasional blood streaked sputum May result from alveolar rupture following sudden exertion in advanced cases | May be the first indication of tuberculous infection May be consequent upon the development of pneumothorax May occasionally be excessive if cavities are present |

*General Complaints.* Weakness, loss of weight, digestive disturbances, night sweats, insomnia, dizziness, and edema of the extremities are not characteristic of uncomplicated silicosis but are apt to be present if infection supervenes, especially when the infection becomes extensive.

## OBJECTIVE SYMPTOMS

Changes in the general appearance are infrequent in simple silicosis unless far advanced. Such changes as are manifested are usually due to complicating conditions

*Silicosis*

Early cases appear unchanged; in fact, it is common to find these individuals showing a *slight increase in weight,* possibly because they are less active As the disease progresses, respiratory embarrassment is noticeable and there is a general loss of muscle tone.

*Silicosis with Infection*

The appearance sooner or later becomes that of chronic phthisis. The bony landmarks of the *thorax become prominent and* there is an increase in the anterior-posterior diameter of the chest, possible hypertrophy of the accessory respiratory muscles of the chest, and in the final stages, retraction of the supra and infra clavicular spaces. Cyanosis and clubbing of the fingers are not prominent except in those cases of long standing, with cardiac disturbances

*Chest Expansion.* Decrease in the expansion of the chest may be demonstrated in cases with extensive pulmonary fibrosis

*Silicosis*

In early cases it is usually not possible to demonstrate de-

*Silicosis with Infection*

Decrease in expansion may not be noted in early silicosis with

creased expansion In advanced cases, expansion may be lessened by 20 to 30 per cent but remains equal on both sides

slight infection but as the condition progresses, a definite decrease is readily observed When infection is more pronounced in one area of the lung, expansion may be more markedly decreased on the affected side, particularly if there is pleural involvement

*Prolonged Expiration* In most cases, decreased chest expansion is preceded and later accompanied by a definite change in respiratory rhythm Close observation reveals that even at rest there is a distinct tendency to prolongation of the expiratory phase, which, as silicosis advances, becomes more marked Following exercise, the silicotic may breathe less rapidly than the normal person under similar conditions as the lungs cannot be emptied rapidly enough to permit more rapid respiration , but although the respiratory rate is not so rapid, as in the normal person, it will persist for a longer period

*Silicosis*

Early in the development of simple silicosis, prolonged expiration may be evident only after exertion Later the degree of prolongation usually parallels the increase in pulmonary fibrosis

*Silicosis with Infection*

In cases of early silicosis with slight infection, prolongation of the expiratory phase may be no more marked than in simple silicosis As the condition progresses and fibrosis increases, it may simulate characteristic asthmatic respiratory rhythm

*Signs Revealed by Palpation of the Chest* Except in the late stages of silicosis with infection, little is revealed by pal-

pation of the chest However, where there is a measurable decrease in expansion, one may note by palpation that, upon forced inspiration, the anterior chest wall is lifted forward by the accessory muscles of respiration.

| *Silicosis* | *Silicosis with Infection* |
|---|---|
| No change noted until extensive fibrosis has taken place, when there may be an increase in tactile fremitus generally | When infection is extensive, tactile fremitus is increased and occasionally friction rubs may be elicited. Extensive thickening of the pleura or pneumothorax may result in a decrease or absence of tactile fremitus over affected areas. |

*Percussion* There is usually an impairment of the percussion noted over the whole chest but unless one is particularly observant, this may not be detected.

| *Silicosis* | *Silicosis with Infection* |
|---|---|
| Since impairment in resonance is general over all lung areas, it is difficult to demonstrate until advanced fibrotic changes have developed Decrease in diaphragmatic excursion may sometimes be revealed by percussion | Increase in loss of normal resonance. When massive areas of fibrosis have developed, this may amount to absolute dullness over areas involved Amphoric resonance may be elicited where there is pneumothorax. Decreased diaphragmatic excursion may readily be shown in advanced infection |

*Auscultation*. Breath sounds are usually decreased in intensity and the characteristic prolongation of expiration is readily noted

| *Silicosis* | *Silicosis with Infection* |
|---|---|
| Decrease in breath sounds general and more marked as the condition progresses Sub-crepitant rales, which clear up after coughing, are occasionally heard | Usually heard, some of the following. Persistent post-tussic crepitant and sub-crepitant rales, coarse rhonchi associated with productive coughing, wheezing or musical rales increased by exertion and coughing, amphoric breath sounds over cavities and areas of pneumothorax: pleural friction rubs occasionally |

## *Diagnosis*

The diagnosis of silicosis must be based upon information furnished by past occupational and medical history, the record of present complaints, and the evidence revealed by a careful physical examination of the chest, including a roentgenogram.

The past and present occupational history should, as far as possible, detail the specific jobs in each industry in which the worker has been employed during his entire working life and estimate the time spent at each job and the duration and nature of previous dust exposure

The medical history should include all disabling illnesses and any history of contact with cases of active tuberculosis The dates of all illnesses should be recorded.

Physical examination of the chest should be carried out in a systematic and thorough manner. Subjective symptoms and positive clinical signs are usually absent or difficult to elicit in early and uncomplicated silicosis, *there are no signs or* symptoms which may be considered as pathognomonic Where the disease has progressed to the point when there is

decreased capacity for work, there will usually be clinical evidence of pulmonary changes.

Included in the diagnosis should be a statement whether there is evidence of tuberculous infection, either active or latent, or infection of other nature.

A positive diagnosis of silicosis should not be made without a good roentgenogram of the chest, revealing nodular shadows throughout the lung fields. Although it is not advisable to make a diagnosis of silicosis based upon roentgen-ray findings alone, nevertheless the chest conditions revealed by fluoroscopic and roentgenological examinations of the chest furnish the most convincing evidence of pulmonary fibrosis Diagnosis by roentgen-ray is discussed in a separate chapter.

A fluoroscopic examination is of distinct advantage. Particularly is it possible, by such procedure, to study diaphragmatic movements and note the presence of areas of increased density which, of course, are better shown in detail by film examination The general information regarding pulmonary density furnished by fluoroscopic examination is of benefit in deciding the particular technique to use to secure the best roentgenogram of the lungs

### *Laboratory Examination*

Laboratory examination of the blood, urine, and sputum are of value when correlated with the clinical manifestations of silicosis. There are no blood characteristic of simple silicosis When infection is present, blood examinations are sometimes helpful in determining its severity and progress Cases complicated by tuberculosis often show an increase in the sedimentation rate, paralleling the activity of the tuberculous complication.

Urinalysis offers little of particular value in silicosis. Uri

nary silica determinations may show an increase in silica present, paralleling the silica exposure [28]

The chief value of sputum examination is to determine the presence of tubercle bacilli As in the case of chronic fibroid phthisis, too much dependence must not be placed upon microscopic examinations of the sputum Where many sputum examinations have been negative for tubercle bacilli, guinea pig inoculation or sputum culture may indicate their presence.

## The Relation of Silicosis to Disability

A diagnosis of silicosis does not necessarily mean disability Since it is a slowly developing disease, roentgenological evidence of silicosis may exist for many years prior to the demonstration of any decreased capacity for work

The term 'disability' is usually employed to indicate lessened capacity to do the work required of an individual in the course of his usual occupation It may vary from partial to complete Disability may persist as partial, but when the fibrosis is complicated by pulmonary tuberculosis, it becomes serious and permanent Individuals with active tuberculosis are considered unsuitable for further employment in even a minimal silica exposure, regardless of whether or not they may also be silicotic

The existence of marked or complete disability is relatively easy to establish However, the clinician may find it possible to arrive at such a conclusion only after repeated examinations, made over such a period of time as to afford him an opportunity to determine definitely the nature and severity of infection and other complicating conditions

The matter of partial disability is far more difficult to determine satisfactorily. The cause and extent of partial disability frequently requires careful examination to be ac-

curately gauged before proper placement of a worker can be effected or a fair settlement of a compensation claim achieved

To declare whether or not a workman should be allowed to continue at his work is a grave responsibility and calls for the utmost care, as well as experience, on the part of the examining physician. Fitness for work may be expressed as follows.

I Partial disability due to silicosis without infection.
 (a) May continue at usual occupation, if environment is satisfactorily controlled as regards dust concentrations and tuberculosis contact;
 (b) Degree of fibrosis and rate of development may require that the individual seek less arduous employment and avoid even minimal exposures to silica-containing atmosphere

II. Partial disability due to silicosis with complicating pulmonary infection
 (a) Primary silicosis Infection mild and non-tuberculous In most instances, after successful treatment of non-tuberculous complicating infection, may be allowed to return to usual work, provided environment under which they are working is safely controlled
 (b) Primary silicosis with pulmonary tuberculosis as complicating infection. Those individuals with silicosis who develop pulmonary tuberculosis are considered totally unfit for further employment in an industry affording even minimal exposures to free silica. This disability is obviously total, so long as active tuberculosis is present. Following successful treatment, these cases regain their health to the point where they can be safely employed at other work.

### Prognosis

There is but a limited amount of factual data regarding the progress of silicosis and the relationship of a previous or superimposed infection to the progressive nature of this condition must be considered Nor is there adequate information as to the ultimate results to silicosis cases after removal from exposure, and the extent of their subsequent pulmonary infection

It may be stated that the prognosis of the individual case of silicosis is almost as variable as are the industrial conditions which cause the disease Where the hazard is not very serious and the resulting damage to the lungs of slight or moderate degree, with removal from further dust exposure, there may be little to apprehend either in the way of disability or shortened life Tubercle infection occurring in such cases tends to become chronic and the patient may live for a long time with no marked disability as do other cases of fibroid phthisis

Where the pulmonary damage from silica is considerable, the outlook is usually unfavorable and when, as so often happens, this type of patient becomes tuberculous, his prognosis is very grave Everything considered, the silicotic who is removed from further dust exposure has a fairly good prognosis When tuberculosis supervenes, he may go the quick route or the slow one. His chances of securing an arrest of his tuberculosis are much less good than if his lungs were not previously damaged by silica

## 2 ETIOLOGY, SYMPTOMS, AND DIAGNOSIS OF ASBESTOSIS

Asbestosis has been defined by Merewether [29] as 'a specific occupational disease of the lungs caused by the inhalation of asbestos dust and characterized by progressive replacement

of the essential active functioning tissue of the lung by inactive non-functioning fibrous or scar tissue. It is essentially a pneumoconiosis, a fibrosis of the lungs, caused by the inhalation of dust and, therefore, is in the same category as silicosis, which disease it resembles in some respects, while differing considerably in others'

Asbestos is a hydrated magnesium silicate. More than 90 per cent of the raw mineral used in manufacture in the United States and Great Britain is Canadian chrysotile. While most of the asbestos dusts in industrial plants are made up of very small particles, asbestos fibers exceeding 200 microns in length have been found in the lung tissue of asbestos workers

*Asbestos is subjected to two main manufacturing processes* It may be combined with diatomaceous earth and other similar inert substances to be used for packing, insulating and fireproofing materials, or it may be combined with cotton or other materials and submitted to the usual textile processes of cleaning, mixing, carding, spinning, twisting, and weaving The asbestos cloth may in turn be subjected to further processes of impregnation with various substances for special purposes

Merewether and Price,[30] in their comprehensive report, state that approximately 2,200 individuals are exposed to practically pure asbestos dust in the factories of Great Britain They examined 363 workers and the following table gives the result.

| *Years at Work* | *Cases Examined* | *Showing Fibrosis* | *Per cent* |
|---|---|---|---|
| 0 to 4 | 89 | 0 | 0·0 |
| 5 to 9 | 141 | 36 | 25 5 |
| 10 to 14 | 84 | 27 | 32 1 |
| 15 to 19 | 28 | 15 | 53·6 |
| 20 and over | 21 | 17 | 80·9 |

This table does not apply to any one group of workers in the factories. The authors have collected also, outside of these statistical cases, records of ten deaths resulting from asbestosis, nine of which were verified by autopsy and the tenth, by repeated clinical and roentgenographic examinations Nine of these deaths occurred from 1928 to 1929. The exposure of the ten cases varied from nine to twenty-four years.

In a study commenced in 1929, Lanza, McConnell, and Fehnel[31] arrived at the following conclusions:

1. Prolonged exposure to asbestos dust causes a pulmonary fibrosis different from silicosis and demonstrable by roentgenogram. Clinically, it appears to be of a type milder than silicosis.
2. Definite cardiac enlargement was frequently found to be associated with asbestosis.
3. A predisposition to tuberculosis, due to asbestos dust, was not indicated although it was not known how much asbestosis may add to the mortality from pneumonia and acute non-tuberculous pulmonary infections.

Page and Bloomfield,[32] of the United States Public Health Service, have given a comprehensive report on dust control methods in an asbestos fabricating plant, and Dreessen, Dallavalle Edwards, and Miller,[33] of the United States Public Health Service, have made a recent study of the asbestos industry This study included physical examinations of 541 asbestos workers and of a control group. No cases of asbestosis were found among workers exposed to dust concentrations below 2,500,000 particles per cubic foot of air. Three doubtful cases were found in the 2,500,000 to 4,900,000 particles range The definite cases were found where the exposure had exceeded 5,000,000 particles The authors suggest 5,000,000 particles per cubic foot of air as a threshold limit pending further investigations.

Carders showed the highest proportionate amount of as-

bestosis, next were mule and ring spinners, and then weavers. As with silicosis, it was found that the incidence of asbestosis increased rapidly with increasing dust exposure. A fifth of those with an exposure of 50,000,000 to 99,000,000 particle years and one half of those with more than 100,000,000 particle years, had asbestosis. No free silica was found in the dust of the factories studied.

### Symptoms

The onset is gradual and depends upon the nature and extent of exposure to asbestos dust. There is a general impression that asbestosis comes on more quickly than silicosis and whereas the roentgenogram may show considerable involvement in silicosis with few, if any, symptoms, the reverse is apt to be found in asbestosis. Cough is variable and expectoration little or none, unless there is an accompanying infection. Anorexia is a fairly constant late symptom and Haddow [34] regards it as an indication to stop work.

As in silicosis, dyspnea is the most striking symptom. It is progressive and severe and is due to impaired elasticity of the lungs and to interference with blood supply. Cyanosis and clubbing of the fingers are common late in the disease as is loss of weight and emaciation

There is a typical skin lesion found in asbestos workers known as the asbestos corn The fine asbestos fibers penetrate the superficial layers of the skin and produce small corns or hypertrophies of the epiderm No asbestosis bodies have been found in these lesions.

### Asbestosis Bodies

Asbestosis bodies are, perhaps, the most interesting feature of pulmonary asbestosis. They have been described in detail by numerous authors, notably Cooke,[35] McDonald,[36] Simson,[37] Gloyne,[38] Merewether and Price,[30] and Lynch and

Smith[39] The description given by Cooke is abstracted as follows:

The 'curious bodies' so characteristic of pulmonary asbestosis are found in the alveoli and bronchioles and in the fibrous and necrotic areas They measure 20 to 100 microns in length One or both ends are bulbous, giving a clubbed or dumb bell appearance The shafts are either homogeneous or segmented crosswise They are golden yellow to brownish in color. They do not stain but give a Prussian blue reaction to iron The Bragg roentgen ray spectoscope showed that they were not altered asbestos fibers Most of them are soluble in strong acids and alkalis and Cooke, McDonald, and Gloyne have been able, by this means, to identify a slender mineral core derived from the asbestos fiber Lynch and Smith[39] were able to see this core as a central filament with slightly greenish tinge These bodies are not essentially iron, but contain it They are not found in the lungs of iron workers

These curious bodies have been induced experimentally Simson[37] states that Mavrogordato gave him for examination the lungs of a guinea pig which had been exposed to asbestos dust from the mines two hours a day for fifty days The animal died from other cause The microscopic examination of lung structure showed a slight generalized fibrosis and the curious bodies similar to those found in the human. Gloyne[18] injected asbestos dust into animals and after twelve days found foreign body giant cells but no curious bodies

From a diagnostic standpoint, it is generally agreed that the curious bodies' signify exposure to asbestos dust but cannot be depended upon for a diagnosis of asbestosis[40]

## Diagnosis

The diagnosis of asbestosis must be predicated on the history of exposure, including the essential factors of length of exposure and its extent, as well as a characteristic roentgeno-

logical appearance, *together with definite symptoms that* cannot be ascribed to other organic disease. Because the pulmonary fibrosis of asbestosis tends to cause cardiac enlargement and impairment, caution should be taken to avoid diagnosing, as asbestosis, cardiac or cardio-renal vascular disease in an individual merely because he worked in an asbestos plant, even though there may be asbestos bodies in the sputum. With the complications of pulmonary tuberculosis or cardiac disease present, the occupational factor may be difficult to determine even by the roentgenologist after a series of films and a post-mortem may be necessary to ascertain to what extent asbestosis, if present, contributed to disability or death. The roentgenological diagnosis of asbestosis is discussed in another chapter.

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## III. ROENTGEN-RAY DIAGNOSIS

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FOR many years, pneumoconiosis, in spite of its often rather spectacular roentgenographic appearances, was very largely regarded in the light of an interesting disorder. More recently, however, the extreme importance of roentgenograms as a means of accurate diagnosis in indicating the exact pathological condition present in connection with pneumoconiosis has received considerable recognition. At the present time, *it is generally conceded that the roentgenographic examination, properly carried through, is the most precise method at our command for determining lung pathology in a case of suspected pneumoconiosis, or for differentiating this affection from another one that may be present.*

The proper roentgen interpretation of pneumoconiosis cases is dependent upon certain important qualifications These may be enumerated as, (1) a knowledge of the anatomy of the chest and the many physiological problems associated with its anatomical constituents; (2) a thorough familiarity with normal roentgenographic and fluoroscopic appearances and their permissible variations therefrom; (3) an understanding of the histology of the lungs and more especially of the lymphatic system; (4) a clear preception of the pathology of pneumoconiosis and of all conditions which may simulate its roentgenographic appearances; (5)

an experienced intimacy with the roentgenographic appearances of pneumoconiosis and of those which may resemble it, based upon a fundamental knowledge of the pathology represented, (6) a keen insight of the physical factors involved in the production of the suspected or alleged pneumoconiosis, and, (7) the use of the proper technic to show, to full advantage, any or all of the abnormalities present. Technic may completely enlighten, may so modify appearances as to be confusing, or may mislead entirely

## The Healthy Chest[1]

Familiarity with the roentgen appearances of the healthy chest is a prerequisite for anyone who attempts to properly interpret chest roentgenograms Unfamiliarity with normal variants and with the modifying influences of a faulty technic not infrequently results in misinterpretation, with an attendant chain of difficulties A common example of such an error is the diagnosis of pulmonary tuberculosis, pneumoconiosis, and silicosis based on the presence of prominent truncal shadows. Within recent years, improvements in certain technical factors and apparatus have created marked variations and changes in the roentgenographic appearances of some of the chest structures. In view of such facts, therefore, a review, and where necessary a revision, of the more pertinent aspects of the chest problem, both in health and disease, seems a necessity

The various tissues demonstrable in the chest roentgenogram will be discussed individually and in the order in which they are taken up in our routine consideration of such an examination To follow a definite system in the study of any roentgenograms is to prevent the overlooking of, in some instances, obvious conditions

*The Soft Parts* — The approximate development of fat and musculature can readily be determined from the roent-

genogram. The density of the axillary fold frequently serves as an index of the patient's state of nutrition A loss, as well as a gain, in weight can easily be determined by comparison of the thickness of the soft parts in those instances in which repeated chest exposures have been made. Sometimes, apparent roentgen evidence of diffuse pulmonary haziness and lung pathology may be explained by unusually thick soft parts Examples of such instances include the well-developed pectoral folds in men, and the large breast shadows in women.

The nipples of the breasts and pigmented moles may produce shadows in the roentgenogram that simulate those of metastatic nodules or other pathological processes. Other soft tissue shadows, such as calcifications of vessels, angiomas, tumors, thyroid, and lymph nodes, may be observed in the neck and axillae Shadows of soft tissue masses (benign or malignant) and enlarged lymph nodes can be demonstrated in the axillae, neck, or supraclavicular fossae

*The Bones.* — The bony thorax can be fairly well visualized in the chest roentgenogram in the anteroposterior, posteroanterior, and lateral projections.

In the *posteroanterior* and *anteroposterior views*, with the sternal ends of the clavicles equidistant from the spine, in order that the chest be symmetrically placed, one can see the clavicles, the upper end of the humeri and the scapulae, the ribs, the cartilages when ossified, and the alignment of the thoracic spine In a few instances, one can see the irregular appearance of the rhomboid fossae on the under surface of the inner fifth of the clavicle This may be unilateral or bilateral and should not be mistaken for a bone lesion Occasionally, one can see the shadow of the foramen of the supraclavicular nerve, near the upper aspect of the middle of the clavicle [2] The epiphyseal development of the inner end of the clavicles can be readily demonstrated, as can epiphyseal centers in the humeri and scapulae. The normal variants of

the ribs, such as bicipital and cervical rib, should be noted. A mental note should be made as to the density of the ossification of the bones. Not infrequently, decreased bone density, caused by some condition such as parathyroid disease, or even localized areas of bone destruction from metastatic malignancy, will assist in directing the attention of the clinician to the primary cause of the patient's complaint. Recognition of erosion of the under surface of the ribs may lead to a diagnosis of coarctation of the aorta. Ossification of the costal cartilages is of very little significance. We place no reliance on the old dictum that ossification is evidence of a chronic bronchitis. In general, ossification increases with age, but occasionally it is found well advanced in thin young women. In such instances, it may be difficult, at times, to interpret structures near the midline, due to the superimposed shadows of the costal cartilages. Oblique views are of great assistance in these patients. There seems to be very little difference in the width of the intercostal spaces in the absence of pleural involvement. In older individuals with increased aeration or emphysema, the intercostal spaces may be slightly wider, but the important manifestation is that the ribs assume a more right-angled position with the spine. The alignment of the thoracic spine can be readily demonstrated. Minor degrees of scoliosis do not interfere with study of the chest, but marked scoliotic changes, on the other hand, tend to obscure the shadows of the structures in both lung fields, due to the shadows of the spine on one side and the closeness of the ribs on the other.

In the *lateral view*, one can see the ribs, portions of the shoulder girdle, and the thoracic spine. The spine is readily visualized but superimposed shadows of lung structures a confusing in evaluating bone detail. In adults, the body o the vertebrae may, or may not show hypertrophic change and even calcification of the nucleus pulposus. Usually the

is a gentle, double curve to the spine, concave posteriorly above and slightly convex, or straight, below. In kyphotic individuals, the anteroposterior diameter of the chest may be greater than the transverse. The vertical diameter of the body of the vertebrae and the intervertebral discs increases slightly from above downward

*The Pleura.* — The parietal pleura is so thin that it does not cast a shadow which can be seen in the roentgenogram unless it is thickened and diseased or obliterates some portion of the costophrenic sulci. Very slight thickenings of the parietal pleura may be suspected in those instances when there is interference with the costal expansion, especially when the process is unilateral

In anteroposterior roentgenograms of the chest made with the Potter-Bucky diaphragm, the reflection of the pleura, ligaments and muscles over the vertebral column produces a straight line shadow on each side of the spine. Disturbance in this normal straight line may occur in spinal caries, bone tumors, some spinal cord tumors that emerge from the spinal canal, and pleural collections occurring in the vicinity of the vertebrae.

In this country particularly, where one finds many references to the apical cap (thickening of the visceral pleura over the apices of the upper lobes), it is a wise roentgenologist who recalls that there are other structures whose shadows create a similar appearance in a similar position. Subcostal muscles, in the oblique views, may be misinterpreted as thickened pleura.

The roentgen appearances of these various anatomical structures have been emphasized by Knutsson,[3] whose observations are briefly abstracted. On the posterior portion of the thoracic wall, the internal surfaces of the ribs are covered with subcostal muscles. In the lateral aspect of the thorax, the muscle lining disappears The narrow muscle layer on

the inner aspect of the ribs produces a shadow, seen only in the oblique view, with one exception, which gets thinner from above downward and disappears entirely in the region of the costophrenic sulci. In pleurisy, on the other hand, the pleural thickening usually gets thicker from above downward,

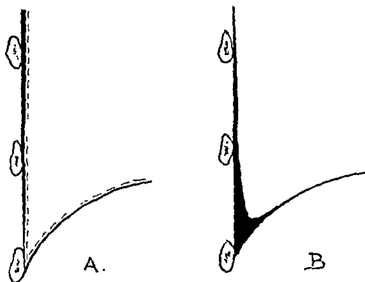



FIGURE 1

and may obliterate the sulcus. (Figure 1) The thickness of the muscle shadow depends upon the muscular development of the individual. In the upper thorax, the muscles extend laterally, far enough to be seen in the posteroanterior and the anteroposterior projections. This structure is called the 'companion shadow of the second rib.' Mesial to the angle

of the second rib, there are no subcostal muscles, and the shadow here is produced by the connective tissue. The first rib, likewise, has a companion shadow which is produced by a sheet of connective tissue interposed between the posterior part of the dome of the pleura and the first and, to a certain degree, the second rib The *following structures are situated* in this interspace , the last cervical ganglion of the sympathetic, the supreme thoracic artery and vein, and the first *thoracic nerve*. The 'companion shadows of the first and second ribs,' as well as those produced by the subcostal muscles in the oblique views, are symmetrical on the two sides This fact should be of assistance in differentiating these shadows from those created by pleural thickening. The pleura does not calcify under normal circumstances.

The visceral pleura covers the different lobes of the lungs and ordinarily does not cast a shadow that can be seen in the roentgenogram, except in the instance of the azygos and other accessory lobes At times, straight and very thin shadows of the interlobar lines can be seen in the chests of healthy individuals We have felt that such pleural shadows represent a permanent thickening of the pleura from a pre existing infection. Furthermore, such slight thickening of the interlobar pleura is capable of producing shadows because the roentgen rays are projected in their axial plane This conclusion is supported by the frequency with which a shadow of the interlobar pleura is found in one roentgenogram of a stereoscopic pair and not in the other. Any interlobar thickening not observed in an axial plane will, nevertheless, produce *shadows in the roentgenogram*, which, when superimposed upon those of the lung fields, will create an increased density If such a density is not recognized in its true relation, it may be *mistaken for an intrapulmonary lesion* In disease, the visceral pleura rarely, if ever, becomes as thick as the parietal pleura. Clark [4] believes that, when adhesions have

formed, the increase in the thickening of the pleura in the lower chest and around the heart, as compared to that occurring in the upper chest, is due in part to the relatively more powerful movements of structures such as the domes of the diaphragm, the flaring of the ribs, and the pulsation of the heart. The visceral pleura is reflected over the mediastinal structures and does not cast a recognizable shadow. In disease, however, it may produce changes which can be observed in the roentgenogram

*The Mediastinum* — In roentgenograms of the chest, we prefer an exposure that will show the shadow of the trachea down to, and including, its bifurcation. In the posteroanterior or anteroposterior roentgenograms, with the patient placed correctly, the shadow of the trachea in the neck will be seen in the midline Deviation to the right or left of the midline above the level of the clavicles may result from rotation of the head, and must be differentiated from displacements of other origin. Below the clavicles, the trachea continues in its midline position until it reaches the level of the aorta, where it deviates slightly to the right in roentgenograms exposed during the inspiratory phase of respiration. In adults, in roentgenograms made in the expiratory phase, the trachea is displaced slightly to the right throughout its thoracic position In all instances, the shadow of the lumen of the trachea is fairly uniform, although its lumen decreases slightly from above downward Just above the trachea, the laryngeal portion of the upper respiratory pathway is narrowed and produces a funnel-shaped shadow, with the apex of the funnel cephally placed

The tracheal cartilages, especially when ossified, aid greatly in the demarcation of the trachea [5] Careful inspection of the shadow of its contour will not infrequently show evidences of compression or deviation that may prove significant. As a rule, the lumen of the trachea is larger in men than in

women. Its caliber is not uniform throughout, becoming progressively narrower as it approaches the bifurcation.

The level of the bifurcation of the trachea varies with age In adults, it usually divides at the level of the intervertebral disc between the fourth and fifth, or as low as that between the fifth and sixth, vertebral bodies It is well to remember, however, that the relationship in the cadaver may vary considerably from that in the living. Likewise, the level of the roentgen tube may cause discrepancies in the level of the bifurcation and the carina.

The angles made by the bifurcating bronchi with the trachea vary with age and from one another. In adults, the angle of the right bronchus is approximately 20 degrees and the left, 40 degrees. The eparterial bronchus sometimes branches directly from the trachea.

The shadows of the trachea and the right and left bronchus can readily be seen in the oblique and lateral projections of the chest. In the lateral view, the trachea may be followed from the suprasternal region down to its bifurcation. During inspiration, the entire trachea moves downward and slightly forward, unless fixed by mediastinitis.

The soft tissues flanking and surrounding the trachea have been discussed by Dann [6] His investigators have helped substantially in identifying the structures producing these shadows The right border of the supracardiac portion of the mediastinum is formed by the superior vena cava and innominate artery, while the most lateral margin of the mediastinal shadow is almost invariably formed by the superior vena cava. ( Figure 2 ) The more medial density is a combination of the vena cava and the ascending aorta, which lies slightly anteriorly and to the left.[7] The space between the vena cava and aorta, as described by Dann [6] in his studies in young cadavers, is rarely seen roentgenographically. However, a markedly distended aorta may approach the right border of




FIGURES 2 AND 3 Healthy adult chest *a* superior vena cava, *b* trachea, *c* hilum or root zone; *d* pre-aortic space, *e* a composite shadow, made up of the inferior vena cava, the phrenopericardial ligaments, and possibly also fluid in the cul-de-sac

the superior vena cava and, in some instances, actually forms the right border of the superior mediastinum. When enlarged shadows of this general configuration are seen, one should consider a substernal tumor, aneurysm of the innominate artery, and diverticulum of the esophagus. Roentgenoscopic observations of the changes in the shadows during the grunt of forced expiration and a study of the esophagus may assist in differentiating between substernal thyroids, innominate aneurysms and esophageal diverticula.

The left border of the supra-aortic portion of the superior mediastinum is formed by the soft tissue shadows of the left common carotid and subclavian arteries. The recurrent laryngeal nerve lies to the left of the trachea. The shadow of the aortic arch is usually seen extending beyond the left border of the spine. With increasing age, its knob-like contour becomes more prominent, until it is most pronounced during the arteriosclerotic period of life. The left posteroanterior oblique view of the chest permits excellent visualization of the aorta and, when used in conjunction with roentgenoscopy, gives valuable information concerning aortic dimensions and elasticity. The aorta follows a more sagittal course in narrow-chested individuals than in flat-chested persons. Increases in prominence in the outline of the shadow of the aortic knob may result from syphilis or hypertension, while an absence or decrease in the outline of the knob suggests either coarctation or right aortic arch.

The pulmonary artery is seen best with the patient in the anteroposterior or right antero-oblique positions. It lies just to the left of the base of the aorta, nestled within the confines of the aortic arch. Sometimes it is seen better after a cough, which serves to separate it from the aorta.[3]

In recent communications, O'Kane, Andrew and Warren[8] and Margolies[9] reemphasize the importance of the roentgenographic and roentgenoscopic examinations in determining

the size and shape of the heart In addition to these estimations, the roentgenologist must concern himself with the pulsations and the mobility of the heart and any abnormal densities There are many conditions, such as congenital deformities of the chest and acquired affections, such as pneumothorax and hydrothorax, which may exert profound influences upon the heart. Examinations must be made in the anteroposterior, posteroanterior, oblique, and lateral views, in order to assure adequate study of the heart The most important view is the left antero-oblique view, but the degree of rotation and the optimum position for a roentgenographic demonstration must always be guided by the preceding roentgenoscopic impressions

Under normal conditions, the heart in thin individuals lies in the vertical position, whereas in the more stocky, broad-chested person, it assumes a more transverse position. Furthermore, patients with round chests more frequently have a rounded heart than patients with flat chests.[10] In the latter type of patient, the heart has a more oval circumference.

Normally, with the patient facing in an anteroposterior direction, the right border of the heart is formed, from above downward, by the right auricle, the inferior vena cava and the cardiophrenic ligament.[9] (Figure 3) In some patients, paracardial fat pads form part of the cardiac border Roentgenographically, however, the shadow of the outline of the heart may sometimes be seen as a density within the less dense fat pad The left cardiac border is formed by the pulmonary artery, the left auricle, and the left ventricle [9] With the patient in the lateral position, the anterior border of the cardiac silhouette is formed, from above downward, by the base of the aorta, the pulmonary conus and the right ventricle The posterior border is formed, from above downward, by the arch of the aorta, the pulmonary artery, the left auricle, the left ventricle and the inferior vena cava The cardiac silhouette,

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with the patient in the right anterior oblique position, is formed by the right auricle and the inferior vena cava on the right side, with the aorta, the pulmonary artery, the pulmonary conus, and the right ventricle, mentioned in order, from above downward, forming the left border. With the patient in the left anterior oblique position, the right border is formed by the superior vena cava, the right auricle and the right ventricle, whereas the left border is made up of the silhouette of the aorta, the pulmonary artery, the left auricle and the left ventricle, mentioned in order, from above downward These cardiac contours bear a definite relationship both in size and degree of expansion with one another.

Normally, the average anteroposterior diameter of the heart is about two-fifths of the transverse diameter of the chest [10] While empirical judgment of the size of the heart is used constantly to decide increases in cardiac size, it is by no means satisfactory. The cardiothoracic ratio, diagonal measurements and cardiac area determinations are much more satisfactory. As a rule, however, the average anteroposterior diameter of the heart is less than three-fourths of the average transverse diameter of the heart. Realizing that the average anteroposterior diameter of the heart in females is 8.7 cm. and in males, 7 8 cm., one can obtain a fair impression as to size [10] The oblique diameter of the heart is about one-third larger than the average anteroposterior diameter.

In the lateral view, the attachment of the pericardium may be seen extending down from the posterior edge of the heart shadow. (Figure 3) It is seen as a faint shadow with its posterior border straight, or slightly concave, extending to the superimposed diaphragmatic shadows This shadow is a vantage point for observing slight pericardial effusions,[11] which tend to distend the cul-de-sac and distort its normal concave appearance

During deep inspiration, especially in tall, thin persons,

the right and left phrenopericardial ligaments exert a pull upon the domes of the diaphragm, by virtue of their attachments to the diaphragm, which is synchronous with cardiac pulsation. As this frequently occurs in healthy individuals during deep inspiration, it must not be confused with the abnormal diaphragmatic tug produced by adhesions between the domes of the diaphragm and the pericardium, which may be seen during the expiratory phase of respiration also. Chances for error are minimized by roentgenoscoping patients in the horizontal posture, the patient lying on the right side and the rays projected anteroposteriorly. This allows the mediastinum to drop toward the right, permitting the examiner to see more of the left dome of the diaphragm and its connections with the heart. Phrenopericardial tugging observed in this position may be evidence of adhesions. With the patient lying on one side, the entire mediastinum shifts to the lower side. This evidence can be recorded when the rays are projected in a horizontal plane.[12]

The horizontal position may also be used to advantage in identifying pericardial effusions. One must be well acquainted with the normal appearances of the heart and aorta in this position before using it for refinements in diagnosis. In this position, the transverse diameter of the heart is larger than in the erect position and the width of the shadow of the aorta is greater. This may be due partly to the elevation of the diaphragm. Roesler[13] thinks that there is also an actual increase in the cardiac volume in the horizontal position. Occasionally, a swallow of barium, with the patient in the horizontal position, will outline a cardiac or aortic aneurysmal dilatation, which might otherwise be overlooked.

Under normal conditions, the space between the pericardium and epicardium is very small. It was our good fortune to examine a healthy student with air in the pericardium, as a result of an accidentally inflicted wound. The space between

the pericardium and the epicardium, as outlined by air, was almost negligible when compared with the pneumopericardiogram of tuberculous pericarditis.

While practically every portion of the heart may become calcified in disease, the calcification associated with old age must be considered physiological. In the past, roentgenologists constantly overlooked calcifications within the heart.[14] Calcified valves may be seen in the anteroposterior view near the middle or lower half of the heart The aortic valves are usually seen overlying, or just to the left of, the spine and are, therefore, more adequately seen on slight rotation. Mitral calcifications, however, lie well to the left of the midline, and when seen in the lateral projection, are situated more posterior than the aortic calcifications Roentgenoscopically, these shadows have to and fro movements, going toward the apex during systole and returning during diastole.

*The Diaphragm* — The right dome of the diaphragm is about 1 5 cm above the left dome, in most instances. When transposition of the viscera occurs, the opposite obtains In certain other instances, the shadow of the left dome of the diaphragm is either on the same level or above that of the right dome We presume that, in the absence of eventration or herniation, such instances may be regarded as normal variants The splenic flexure of the colon is usually highly placed in such individuals, or there may be a large collection of air in the fundus of a cascade type of stomach, which may be responsible, to some degree, for the position of the diaphragm

In the anteroposterior and posteroanterior projections, the right and left halves of the diaphragm are dome-shaped. The attachment of the mesial portions of the domes is on a higher level than its attachment to the ribs This results in a costophrenic sulcus of varying depths The domes of the diaphragm are regular in this plane and, during inspiration, any peaking may be the result of either pleural adhesions or

localized areas of inelasticity of the lung structures The domes of the diaphragm may be wavy at their outer aspect or produce a shadow of multiple arcs over its surface, due to variation in attachment or hypertrophy of the constituent muscle bundles. This has been described as individualization of the costal components. Occasionally, in the posteroanterior and lateral projections, one finds a rounded hump of the mesial aspect of the right dome of the diaphragm This observation has been reemphasized [15] as being due in the healthy individual to a weakness of the anteromedial portion of the diaphragm as compared to that of the posterolateral portion In addition, the inferior vena cava may play a part in preventing downward excursion of the diaphragm Tumors of the liver and subphrenic lesions may produce an appearance that simulates such a condition

An anomalous enlargement of the right lobe of the liver will produce a shadow in the lateral view that may simulate that of an interlobar collection [16] In the lateral view, the domes are higher anteriorly and the posterior costophrenic sulcus is much lower than the anterior, rendering it possible for a relatively large localized pleural collection to escape detection in the dorsoventral roentgenogram The upper surface of the shadows of the right and left domes runs from before backward These shadows may be parallel or may cross each other, either anterior or posterior to a point that corresponds to the middle of the domes During the inspiratory phase of respiration, the shadows of the domes lose their dome shape and become relatively straight

The movement of the domes of the diaphragm is extremely variable The most correct impression of its movement is obtained during ordinary respiration. Frequently, efforts to study diaphragmatic excursion during forced respiration are confusing, due to the influence of costal and abdominal muscles The movement of the domes of the diaphragm is usu-

ally more pronounced in men than in women. The domes are higher and the excursion is greater in the horizontal posture than in the erect. Therefore, anyone interested in detecting slight restriction in movement should examine the patient in the erect posture. Lateral roentgenoscopy of the diaphragmatic domes, with the patient lying on the back and the rays projected from the opposite side, is difficult because of the increased dome shape of the diaphragm and the encroachment upon the costophrenic sulci. If the patient is studied roentgenoscopically in the horizontal plane while lying on one side, and the rays projected ventrodorsally, the dependent diaphragm will become more cephally placed and will have a greater excursion. These observations have also been noted by Pierson and Newell.[12] Such positions or modifications thereof allow a study of the costophrenic sulci and aid in differentiating between thickened pleura and pleural collections

*The Hilum Shadows* — The structures in the hila include the bronchi, the pulmonary arteries and veins, the tracheobronchial and aortic lymph nodes, nerves, and connective tissue The lymph nodes are classified as follows; (*a*) bifurcation nodes, located between the right and left main bronchi, (*b*) tracheobronchial nodes, situated above the bronchi and along the trachea; (*c*) the aortic nodes; and (*d*) the paravertebral nodes. Sukiennikow[17] states that there is a greater number of lymph nodes on the right side. Miller[5] says that there is a greater number of nodes associated with the eparterial bronchus than with the bronchi supplying the middle and lower lobes.

The hilum shadow is irregular in outline with ill defined borders The density of these structures, as seen in the roentgenogram, varies considerably with the type of roentgenographic exposure In the arbitrary properly exposed roentgenogram, the shadow has varying densities and is not homo-

geneous. It would seem that if roentgenologists could agree upon what a properly exposed film consists of, varying densities could be regarded as significant observations We have placed considerable reliance upon individual ability to see varying densities in the hilum shadow, i e., the bronchial shadow is different from that of the vessels When the density of the shadow is more or less homogeneous, it is our impression that one can be fairly certain that abnormal changes have become manifest

The National Tuberculosis Committee [11] has described the limits of the hila as follows ·

'On the right side, the normal shadow may be said to extend, from above downward, from the top of the right main bronchus to approximately a point where the right lower lobe bronchus, below the origin of the middle-lobe trunk, begins to subdivide into its lower-lobe branches ( or trunks ) This includes the anatomical hilum, the greater portions of the larger bronchi, and some lung tissue in front and behind The extent of this hilum shadow from above downward is approximately two interspaces and a rib

'On the left side, the pulmonary artery passes above the left bronchus, and from above downward, the left hilum shadow may be said to extend from the upper border of the pulmonary artery down to approximately a point where the left lower lobe bronchus begins to subdivide into its lower-lobe branches ( or trunks ) Such a hilum shadow on this side is slightly shorter than the one on the right and is slightly less than two interspaces and one rib in length

'While the top of the right bronchus is higher than the left, if the tops of the hilum shadows be limited in the manner just described, they are on a plane approximately level

'The thickness of the hilum shadow in the anteroposterior direction is a matter that requires further study, and prob ably largely from the lateral view Measurements from lateral

views are very uncertain and subject to considerable personal equation. One should learn to acquire the idea of depth as well as length and width when interpreting hilum shadows.'

In the lateral view, the shadows of the hila are superimposed and it is impossible to define their limits, even within wide margins, because of so many variable factors, such as target film distance, length of time of exposure, and disturbed shadow outline due to transmitted pulsation from adjacent vessels We suggest, however, that one can develop an individual impression as to their normal extent by studying roentgenograms of the chest of healthy individuals, made with a Potter-Bucky diaphragm in an almost lateral projection, with the patient so placed as to prevent the superimposition of the hila Such films allow one to see shadows of the varying densities of the hila, and, if a thorough understanding is obtained, it becomes possible to determine deviations from the normal with a high degree of precision.

In the dorsoventral and ventrodorsal roentgenograms, the outer boundary is regarded as normal when it lies within the inner zone of the lung field Everyone familiar with the interpretation of variations of hilum shadows realizes how difficult it may be. Oblique and lateral views often assist in detecting glandular enlargements that might otherwise escape notice

Calcified lymph nodes occur frequently in the hila of healthy individuals. They represent, as a rule, evidence of a pre-existing active tuberculous process. Calcification of a lymph node does not necessarily mean that the node may not contain viable tubercle bacilli which, under certain conditions, are capable of multiplying and setting up an active disease. In adults, we have felt that nodes that are calcified in a relatively homogeneous manner are probably healed, whereas those in which the calcification is punctate and appears as a mottled shadow have a much greater potentiality



FIGURE 4

Calcified tracheobronchial lymph nodes. Note the irregular calcification of the nodes. They are somewhat punctate in type. These are examples of nodes in which infection may become reactivated.

for reactivation. (Figure 4) Chests of children with calcified nodes of this type should probably not be regarded as healthy. While it is true that calcified nodes are usually regarded as evidence of past tuberculous infection, it is entirely possible for them to be due to some other process.

It is important to evaluate the dense, circular shadows, due to vascular trunks obtained in an axial plane, and appearing in the roentgenogram as densities similar to those cast by calcified nodes These shadows are readily seen roentgenoscopically and, should any doubt arise as to their nature, slight rotation of the patient will permit identification of their true nature. Calcified shadow densities do not disappear on rotation, whereas vascular shadows do.

*The Zones.* — The chest has been variously divided into different zones for convenience of description of the single film The classification in most frequent use is that which divides the one side of the thoracic cavity into 3 zones; the inner or hilum, the middle or truncal (or vascular markings), and the peripheral, which contains the finer subdivisions of the vascular tree in which the only shadows visible consist of fine linear markings.

In stereoscopic films, there is very little occasion to use such a classification, except so far as the various zones have different capacities for expansion [18] The hilum zone expands and contracts less than the truncal or vascular zone, and the peripheral zone has more expansile and contractile power than the truncal or vascular zone.

*The Lobes* — Ordinarily, the right lung is divided into 3 lobes and the left, into 2 Not infrequently, there is an accessory lobe on the right side called the azygos lobe [19] This lobe is recognized by the fine pleural line which extends from the apex of the lung downward, to end as a dense oval shadow near the upper aspect of the right border of the superior mediastinum The dense oval shadow is *due to the*

azygos vein, which maintains its foetal position and carries with it a reflection of the parietal pleura, down to the point of entrance into the superior vena cava. The size of the azygos lobe is quite variable, depending upon the position of the vein.

More recently, other accessory lobes have been described in roentgenologic literature.[20] An accessory lobe may occasionally be found at the base of either lung, either partially or completely formed. These lobes are variable in size and represent a division of the lower lobe. They can be recognized by the presence of interlobar pleural shadows, extending upward from the mesial portion of the domes of the diaphragm toward the hila. This lobe may occasionally be the site of a pathological process and, therefore, readily recognizable. On one occasion, we observed two lobes only, on the right side. The observation was later confirmed by post-mortem examination.

The position of the various lobes is readily observed in those instances where either interlobar shadows are visible or after introduction of an opaque medium into the bronchi. It should be emphasized, however, that the size and extent of the lobes are quite variable. On occasions, the lower lobes may extend very high posteriorly, so high that physical signs, and even shadows in the roentgenogram, may be misinterpreted as representing lesions in the lower portion of the upper lobe.

*The Vascular Markings.* — Extending from the hilum on either side are heavy shadows passing into each lobe. These shadows are more pronounced in the lower lobes and, in roentgenologic literature, are described as truncal shadows. It has seemed to us, however, that such a terminology conveys an impression that a bronchus is responsible for, or at least associated with, these visible shadows. From the work of Miller,[21] Miller[22, 23, 24, 25, 5] and Greineder,[26] such an inter-

pretation may not be true. For years, roentgenologists attempted to localize foreign bodies and pathological lesions according to the distribution of these shadows as they passed into each lobe. It was recognized that only very large bronchi were visible, and these were restricted, for the most part, to the hilum zone. On the other hand, the roentgenologist thought that the truncal shadow, as seen and described in the roentgenogram, consisted of bronchus, artery, vein, nerves, lymphatics, and connective tissue. Wherever one of the large branch shadows was seen, there was thought to be a bronchus We know now that such is not the case Miller[23] has shown that 'the pulmonary artery follows, in all of its subdivisions, the subdivisions of the bronchial tree. As each main branch of the pulmonary artery arches over its corresponding stem bronchus, it comes to occupy a position posterior (dorsal) and slightly lateral to the bronchus. The relation of the main trunks of the pulmonary vein to the bronchi is quite different, they are situated anterior (ventral) and mesial to their stem bronchi and in their ultimate distribution are situated as far removed from bronchi as possible'

Miller,[21] interested in another phase of the same subject, made similar observations, as did Greineder,[26] with even more striking demonstrations by tomography. If one can be certain that veins, as a rule, have a larger diameter than the arteries, it is our belief that the shadows of the larger veins can be differentiated from the larger arteries in roentgenograms of excellent detail made during the diastolic phase of the cardiac cycle

We appreciate that the above discussion may be considered academic and of no practical importance so far as the distribution of the bronchi is concerned, because of the improved methods for delineating bronchi after the instillation of opaque media and the increased use of the bronchoscope. Nevertheless, it is our firm belief that too many roentgenolo-



FIGURE 5

Illustration of a roentgenogram showing the vascular markings in the right upper lung field. The pulmonary artery and vein were ligated in this instance. In the left upper lung field, the blood has been allowed to drain out. Note the difference in size of the vascular markings. (Reproduced by permission from Miller[21])

gists are misinterpreting normal variations of the vascular markings in terms of pathology. (Figure 5) Miller,[23] in 1919, recognized and warned us of such a possibility. Now, it has become necessary for industry to have pre-employment as well as follow-up examinations and, unfortunately, many roentgenologists, upon whom the burden of the roentgen examination has fallen, are, *all too frequently, misinterpreting vascular shadows as fibrosis, and thereby imposing an unnecessary hardship on both the employee and the employer.*

Vascular shadows are otherwise confusing in interpretation, particularly with regard to the dense, circular shadows, due to blood vessels recorded in an axial plane, which may simu-




FIGURE 6

Curved paraffin cylinders in *c* were examined to demonstrate differences in appearance when rays were directed in one plane, *a* and in another plane, *b* Note the differences in density of the cylinders in *a*. and *b*. *d* vessel shadows in roentgenogram of inflated normal lung (Reproduced by permission from Miller.[21])

late an appearance of thoracic calcium [21] and, in the more peripheral portions of this zone, may produce an appearance of beading or early nodulation, when the roentgenogram is made during the systolic phase of the cardiac cycle. The appearance of beading may simulate that of a pathological process, such as that seen in silicosis, baritosis, or a mycotic infection. Stereoscopic dorsoventral films may not be sufficient to identify the exact nature of the shadows, but films exposed in an oblique view and rotation of the patient during the roentgenoscopic examination will usually be of aid. If the dense shadow is due to a blood vessel, slight rotation will cause the shadow to disappear, if due to a calcified lesion, the shadow will remain. Furthermore, the blood vessel shadows are circular and homogeneous, whereas a calcified lesion

may be irregular and less homogeneous, or even mottled (Figure 6)

Miller[5] called attention to another structure, the ring-shaped cartilage which occurs where bronchi leave the main stem bronchi. This cartilage may produce a dense, circular shadow in the roentgenogram

McPhedran and Weyl[27, 28] have emphasized the importance of synchronizing the roentgen exposures of the chest during the late diastolic phase of the cardiac cycle. They have performed important experiments in an effort to evaluate this phase of the general problem, but further investigation is necessary. This work, thus far, serves to show that vascular displacement and vibration cause considerable blurring of roentgenographic detail. Likewise, kymograms illustrate clearly what a marked influence the cardiac pulsation exerts on the lung structures close to the heart.

*The Bronchi* — The first subdivision of the right bronchus is the *right upper lobe bronchus* (eparterial), which comes off at an angle of almost 90 degrees and passes above the pulmonary artery This bronchus gives off a branch, the vertebral, which passes upward to the apex of the lung parallel to the spine This bronchus gives off branches which extend anteriorly and posteriorly The upper lobe bronchus gives off another important branch which is designated as the second interspace bronchus, because it comes off behind the level of the second interspace in front. Likewise, this bronchus gives off anterior and posterior branches

The portion of the right bronchus between points from which the upper and middle lobe bronchi come off is termed the *stem bronchus* Foreign bodies frequently lodge in this region

The *middle lobe bronchus* comes off the stem bronchu anteriorly It divides into many branches, but there are main divisions, as a rule, (*a*) the branch which passes dow

ward and anteriorly, and (*b*) the branch that passes upward and laterally.

The *lower lobe bronchus* bifurcates into several main branches which have been variously described as the anterior, middle, posterior division branches in the lower portion of the lower lobe, and the apical branch in the upper portion. All of the main subdivisions give off many branches. Disease of the mesial and posterior division bronchi produce shadows that border upon the shadow of the heart in dorsoventral roentgenograms Further identification of the bronchi can be obtained in the oblique and lateral projections

The *left upper lobe bronchus* (hyparterial) passes below the pulmonary artery. Its subdivisions are similar to those of the right side, except that there is a branch which goes to the lingual tip of the upper lobe which is that portion in front of the heart.

The *left lower lobe bronchus* has a longer course than that of the right side before it gives off any subdivisions, otherwise it and its subdivisions are similar to those of the right side

*The Lung Fields.* — Medicine owed a debt of gratitude to William Snow Miller whose life has been dedicated to the study of the anatomy of the lung. No discussion of the healthy lung can possibly be complete without frequent reference to Miller's work, and much of that which we shall discuss has been gleaned from his reprints and recent book [5]

In the discussion of the healthy lung, we have approached its consideration from the standpoint of the smallest functioning unit in the lung, the primary lobule. It is obvious, therefore, that the following discussion is of more importance histologically than roentgenologically.

As the bronchial tree is followed to its ultimate divisions, there occurs a lack of the regular bronchial outline and the gradual appearance of an air passage, whose walls are lined

with variable, irregularly placed air spaces These terminal air channels are called the respiratory bronchioles, which, in turn, divide into smaller branches known as the alveolar ducts These alveolar ducts are smaller than the respiratory bronchiole from which they spring and are lined by more alveoli than the aforementioned bronchiole [22, 24] The alveolar ducts, in turn, lead into from 2 to 5 spheroidal cavities called atria The atria, in turn, open into numerous air sacs around the periphery of which are situated the alveoli

The bronchial musculature forms sphincters about the channels leading into the atria and around the alveoli which line the smaller bronchioles [25] These sphincters probably play an important role in the muscular spasm known to occur in asthma. Normally, air passes unimpeded by these sphincters With muscle contraction, the air channels reduce in size preventing the egress of air, which produces an increase in residual air with the formation of emphysematous lobules

The structures described above comprise the anatomical unit or primary lobule of the lung, which may be defined as a ductulus alveolaris, the air spaces connected with it, and all their associated vessels, nerves, and lymphatics The primary lobule has the form of a truncated pyramid, with its base directed toward the periphery of the lung [22, 24] Its air passage (ductulus alveolaris) usually enters the primary lobule at the side rather than at its apex Primary lobules vary in size from 0 45 mm to 0 815 mm, the larger units situated in the peripheral and basilar portions of the lung It is obvious that these lobules are not seen in the roentgenogram It is only barely possible that one sees, roentgenographically, the larger, secondary lobules, which are made up of 50 to 250 of these primary units

The primary lobules receive relatively little blood from the bronchial blood vessels [22, 24] The pulmonary artery, on the other hand, follows the course of the respiratory bron-

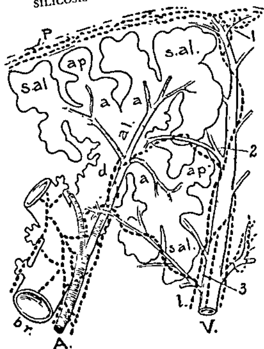



FIGURE 7

Composite diagram (modified from Miller) of the primary lobule lymphatic system, indicating the primary distributions or accumulation points for dust which will lead to predominant phases of pneumoconiosis, b r., respiratory bronchiolo, d al, alveolar ducts, a, atria, s al, sacculi alveolare; a p, alveoli, a p, aveoli opening into respiratory bronchioles and alveolar ducts in close relation with the origins of peribronchial and perivascular lymphatics, A, branch of pulmonary artery, accompanying the air passages; V, branch of pulmonary vein in interlobular septum, P, pleura, P B, peribronchial lymphatics; P.V, perivascular lymphatics, 1, 2, 3 and other dotted areas, lymphoid deposits (By previous permission of Dr W S Miller)

chiole and the alveolar duct into the center of the primary lobule.[5] Each atrium is supplied by a branch of the pulmonary artery, which then divides into enough smaller tributaries to supply the alveolar sacs. The pulmonary vein, in contra-

distinction to the pulmonary artery, courses along the periphery of the primary lobule at a distance from the air channels [5] (Figure 7) Its branches arise from the pleura, the end of the alveolar duct, the walls of the alveolar sacs and the points of division of the bronchi and bronchioles

The lymphatics of the lung may be divided into the deep lymphatics and the superficial lymphatics [24] The deep lymphatics include all of the channels which follow the bronchial tree and its associated blood vessels toward the hilum The superficial lymphatics lie within the pleura

The bronchial lymphatics in the primary lobule are 3 in number, with no lymphatics present in the air spaces as vessels. The lymphatics of the pulmonary artery lie between the artery and the bronchiole, anastomosing with 1 of the 3 bronchial lymphatics These, in turn, unite with others to form a rich network running toward the hilum. The lymphatics of the pulmonary veins follow the course of the veins from the various points of origin described previously, with one exception Since no lymphatics lie in the walls of the air sacs, there are no lymphatics along the veins arising from their walls

The pleural lymphatics form a rich network which drains into the lymph nodes in the hilum. There is some evidence supporting the belief that the superficial pleural lymphatics, covering the diaphragmatic surface of the lower lobes, drain into the abdominal pre-aortic nodes by way of the ligamentum pulmonale [24] The numerous valves in the superficial lymphatics point in no particular direction The valves are less numerous in the deep lymphatics and lie close to the hilum in the large vessels, allowing flow in the direction of the hilum only Similar valves lie in some of the deep lymphatics close to the pleura These valves point toward the pleura, allowing lymph flow into the superficial pleural lymphatics without permitting a reflux of superficial lymph into the deep set of lymphatics

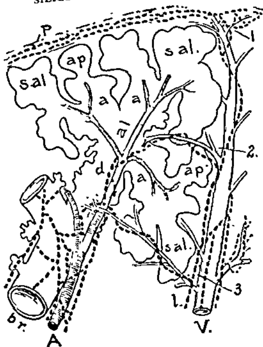



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In addition to the lymph vessels, the primary lobule contains small masses of lymphoid tissues, which may be found in the walls of the primary lobule.[29] Besides these distinct lymphoid masses, one can identify a sheath-like dispersion of lymphoid tissue along the pulmonary artery and along the air channels. This faintly defined sheath of lymphoid tissue is rarely found in the lungs of humans under thirty years of age With the advent of middle life, however, these lymphoid deposits become more conspicuous until they are quite prominent at approximately sixty years of age. The lungs in healthy individuals over sixty frequently show the artery and bronchus completely surrounded by this lymphoid tissue.[29] Perhaps this accounts for the prominent linear markings frequently associated with advancing age, markings which are usually lacking in children and young adults It is important to know the distribution of these lymphatic masses and lymphoid networks as they are the centers about which disease processes often commence

Considerable evidence has been accumulated substantiating the belief that massage, passive motion, capillary wall injury, increased venous and capillary pressure, and decreased blood proteins accelerate lymphatic flow.[30, 31] The importance of these findings may be appreciated when one realizes that the rate of lymph flow and the progress of phagocytes within lymph vessels are negligible when the part being studied is at rest Without the former observations, one would be at loss to explain the rapid appearance in the hilum glands of foreign particles ducted into the lung. In one set of experiments, ink particles were demonstrated within the hilum nodes several minutes after their injection into the lung[30] Drinker and Field[30] demonstrated that phagocytosis was not entirely responsible for the transfer of foreign particles into the perialveolar spaces Perhaps alveolar pores really do exist, allowing free interchange of such material.[32, 33] This, in part,

helps explain the rapid appearance of foreign bodies in the hilum nodes, as the finding obviates complete dependency upon phagocytosis in the transfer of foreign material from the alveolus into the perialveolar tissue

Dolley and Wiese [34] recently found that unilateral pneumothorax produced a decrease of about 15 per cent in the lymph flow from the lungs. Such data, correlated with those previously referred to, would seem to indicate that the respiratory excursions of the lungs and the massaging action of the pleura play an important role in pulmonary lymph flow It also seems reasonable to conclude that the rate of lymph flow might vary in different portions of the lung, depending upon differences in respiratory excursions in various portions of the lung That such differences in the amplitude of pulmonary expansion do occur seems well substantiated by the work of Keith, Miller, Scott, et al

While there is good evidence indicating that lymph constantly courses through the lung toward the hila, no absolute proof concerning the magnitude of this lymph flow is available. Drinker and Field [30] were unable to demonstrate any appreciable flow in their animal experiments If little is known concerning this particular factor, certainly less is known of the true function of the lymphatics

*Effect of the Phases of Respiration on the Roentgen Appearances in the Chest* — During inspiration, the first five ribs rotate up and out, carrying the sternum forward and upward These movements increase the anteroposterior dimensions of the chest, thereby expanding the upper lobe in an anteroposterior direction, allowing an influx of air.[28] The ribs maintain a constant relationship with the upper lobe in its anteroposterior excursions, with the result that the impressions of the first five ribs may frequently be seen in post-mortem specimens on the anterior surface of the upper lobe Such markings, however, are unusual in the lower lobes During inspira

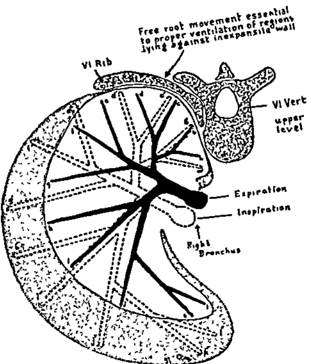



FIGURE 8 See caption on page 99

tion, the lower lobes are expanded by the descent of the powerful diaphragmatic muscles, which increase the depth of the chest, and by the action of the lower intercostal muscles, which increases the transverse diameter of the chest [18] These forces are much greater than those influencing the upper lobes Because of this, the lower lobes are drawn downward during inspiration, thereby massaging its pleural surfaces against the long axis of the ribs, which explains the absence of the rib markings previously referred to as being commonly found in the upper lobes The evidence concerning the formation of rib impressions in the upper lobe is indirect Clark [4] has called our attention to the relative lack of pigmentation of the lung surface where rib impressions are found, whereas more pigmentation is found in the lung tissue adjacent to the intercostal muscles Such evidence is in favor of Keith's [18] ideas concerning the mechanism of respiration Clark [4] also believes that these observations support the premise that the massage effect of the ribs is an important factor in lymphatic flow This seems reasonable in view of the fact that other portions of the lung, such as the lung around the heart, the tip of the lung extending into the costophrenic sulcus ( where it is massaged by the diaphragm ), and the posterior surfaces of the lower lobes, are less pigmented than the inside of the lung and the lung surfaces where massaging action is minimal The summation of these inspiratory forces produces expansion in a forward, downward and outward direction, with considerably more expansion taking place in the lower lobes than in

FIGURE 8

Diagrams showing the differences in expansion in the various portions of the lung *Note decreased expansion in upper lobe and in paravertebral gutter* ( Reproduced by permission from Macklin [36] )

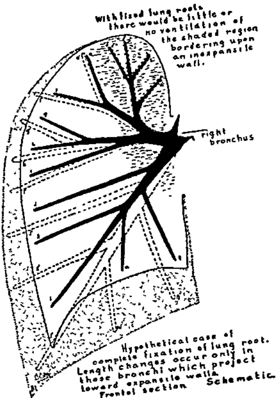



FIGURE 9. See caption on page 101.

the upper [18] The trachea and its ramifications also feel the effects of these inspiratory forces as the trachea and hila move downward and forward, while the bronchial tree and the vascular structures become elongated * [35, 36] Keith [18] has stated that, when more is learned about normal respiration, the lobation of the lung and the great fissures will be found to be of functional significance

Working against these inspiratory mechanisms are the heart, the vertebral column and vertebral portions of the ribs These portions of the chest are antagonists to pulmonary expansion by virtue of their anatomical characteristics [18, 35, 36] Being relatively fixed structures, they cannot move to allow the nearby lung to expand (Figures 8 and 9)

In like fashion, the apex of the upper lobe does not expand as fully as the lower portion of the upper lobe, which is not inhibited by the peculiar conical shape of the upper thoracic cage One can conclude, therefore, that the amount of air entering the lower lung field and periphery of the lung is greater than that which enters the upper lobes or hila

Attention should again be directed to the importance of respiratory movements upon the lymphatic flow and its possible influence upon the distribution of a pathological process While the evidence is purely deductive, it seems reasonable

* Keith and Miller believe that the bronchial tree spreads and elongates during inspiration Macklin's work, while not conclusive does not support the spreading of the bronchi although it does show the elongation very well

FIGURE 9

Diagrams showing differences in expansion in the various portions of the lung in a case with infection The shaded portion of the diagram in the upper, mesial and posterior portion of the lung field represents the area of infection Note the marked decrease in the expansion of the lung Compare with Fig 12 (Reproduced by permission from Macklin [36])

that adequacy of lymphatic flow and degree of respiratory mobility go hand in hand. Perhaps this might account for the frequency of upper lobe tuberculosis and for the mid-lung field changes of silicosis Perhaps it might also account for the clear peripheral and paracardiac zones in the lung fields in patients with pronounced changes due to silicosis with a quiescent infection

*Roentgen Technic.* — Considerable data have been accumulated showing that the diagnostic value of chest roentgenograms depends upon certain film characteristics which are determined by the technical factors used for the examination.[37, 38] For this reason, certain fundamental technical requirements have been defined which, when adhered to, have produced a high quality of chest examination. Briefly, these factors may be summarized as follows:

1 *Time.* Exposures made in from $\frac{1}{30}$ to $\frac{1}{10}$ second are essential to overcome lack of detail resulting from vascular motion Synchronization of the roentgenographic exposures with the late diastolic phase of the cardiac cycle further reduces vascular blurring This method, if coupled with some means of determining full inspiration, would be ideal, as one could then be reasonably sure that slight vascular changes in different films of the same patient were, or were not, due to physiological variations[27, 28]

2. *Distance* Focal spot film distances between 4 and 6 feet are most satisfactory, as distances under 4 feet produce considerable distortion, while exposure made at distances over 6 feet are not sufficiently superior to warrant the expense of bulky high tension apparatus

3. *Screens.* Screens should be chosen for excellence of detail, although high-speed intensifying screens are of considerable assistance in avoiding blurred images. Screens must be kept clean and care taken that contact is maintained throughout their surfaces

4. *Voltage.* The roentgen tube voltage should be so chosen that the densities in the diagnostic areas of the film will lie between the limits of 0.4 and 0.6. This depends upon the other physical factors employed, but, as a rule, will vary from 50 to 80 kv (peak).

5. *Current.* The choice of the tube current also depends upon each of the factors previously considered. Bearing these factors in mind, that tube current should be chosen which will produce proper roentgenographic densities.

6. *The Roentgen Tube.* The line or band focus tubes have been recommended as they combine the detail produced by a small effective focal spot with the durability of the broad focal-spot tube.

7. *Film Processing.* Unless a time-temperature method of development is employed, in which all factors are carefully calibrated and controlled, it is useless to follow the technic described. Warm solutions, impure chemicals, over or under developing will ruin any film, no matter how perfect its exposure may have been.

In addition to these factors, it is necessary to use accurate methods for measuring patients. One must also be careful to make all measurements through the same anatomical points. An impulse timer, a cathode current stabilizer, suitable indicating meters, adequate power supply, full wave rectification for the high tension equipment, and 1 kv per step autotransformer control, are all essential for obtaining roentgenographic results which can be repeated on different occasions.

Stereoscopic examinations made in the anteroposterior or posteroanterior directions are desirable in chest roentgenography. The gross exaggeration of vascular markings and other abnormal densities, seen in single flat films of the chest, is avoided and properly evaluated by stereoscopy, which places these questionable areas at their right depth. Lateral,

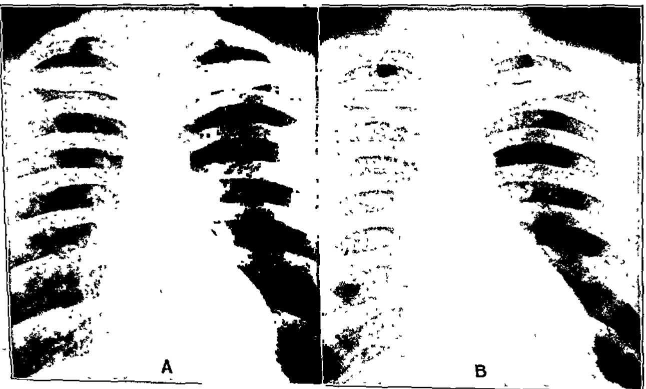



FIGURE 10. See caption on page 105

oblique, recumbent, or any other position, determined roentgenoscopically, will add valuable information

Most chest cassette changers are equipped with some type of fixation device, such as a windlass and binder, which encircles the patient's back for immobilization After immobilization has been accomplished, care must be exercised to determine whether or not the patient is rotated. Even slight rotation will cause a difference in density of the two sides of the chest Such differences in density have been regarded, by some, as evidence of pleural thickening or hypoventilation (Figure 10)

## ROENTGENOLOGICAL STAGES OF PROGRESS [49]

Roentgen appearances represent pathological changes At first, in the study of pneumoconiosis, it seemed logical that the stages of progressive changes should be based upon a sequence of advancement which could be shown by such a simple means as the roentgenogram Clinical groupings must necessarily be based upon such phenomena as increasing dyspnea and incapacity for work, together with certain clinical signs Even these, when the first classifications of pneumoconiosis were based upon similar industries, rather closely followed the roentgenographic demonstration of the progression of pathological changes

It is unfortunate that the roentgenological groupings of the various countries have differed In the United States, the roentgenological classification of Lanza and Childs,[39] who

FIGURE 10

*a* healthy chest, *b* same patient Note the increased density over the right hemithorax which is due to slight rotation This was produced by the supporting strap when the patient was placed against the film changer

divided the condition, as shown by the roentgenogram, into 3 well-known stages, designated numerically, was followed until comparatively recent years, when it no longer seemed feasible. In South Africa, where the disease received probably the greatest amount of study in the early years of our more extensive knowledge of silicosis, three pathological stages were first recognized — early, intermediate and advanced silicosis.[40] The roentgenographic appearances ascribed to these were quite similar to those of our own three stages. Later on, the condition was *legally* divided into three *clinical stages* — ante primary, primary, and secondary. Roentgenographic appearances were grouped to conform to these in showing progression or extent. Compensation laws elsewhere, as in Ontario, have been based, to a considerable degree, upon the highly successful legislation in force in South Africa. As a result, the roentgenological classification of silicosis has followed the same designations of stages as that of the legal classification, which is quite natural. With no federal laws in this country directly governing the condition, and no clinical classification of the disease, the roentgenological classification first adopted has held priority.

In order to make comparisons with the groupings used elsewhere, it is necessary to give a brief résumé of the roentgenological stages of silicosis as described in this country.

*First Stage.* This stage has been characterized roentgenographically by a definite increase in the prominence and extent of the hilum shadows, an increased prominence and thickening of the trunk shadows, and a greater prominence of the linear markings of the peripheral zone. Enlarged glands may be interpreted in the hilum regions. This stage must be subject to certain modifications. Frequently, appearances are detected in the central portions of the lungs, usually more or entirely on the right side, consisting of a slight haze, with or without fine lines. Within these areas,

there occasionally become manifest a few small, faint, hazy spots, which are probably the beginning of lymphoid deposit enlargement In many cases, the definite appearances of the first stage, as just described, are not apparent. In a flat roentgenogram, these hazy areas often have the appearance presented by a very small female breast or a pectoral muscle in a male, but in the wrong locality This manifestation is probably due to the beginning of the interstitial fibrosis and of the visibility of a few of the individual nodules. One who has observed the effects of dusts in many occupations will find that individuals who follow certain dusty trades do not present progressive appearances that run true to form or that can be made to conform to the usual general stages, for the reason that there are types of the condition peculiar to certain industries, and, possibly, to certain individuals

The general manifestations of this first stage are not characteristic of pneumoconiosis alone, but may be simulated by many other conditions, especially, passive congestion, acute or chronic respiratory infections, chronic bronchial catarrh and bronchiectasis Moreover, the hilum and trunk shadows are subject to considerable normal variations in appearance in the adult.

As a numerical stage, this one implies, theoretically at least, an early period in progression, yet individuals may remain in this phase for years, or indefinitely As the manifestations of a typical first stage, with prominent hilum and trunk shadows and linear markings, are not characteristic of pneumoconiosis alone, I am inclined to doubt the fairness of giving this stage a definite medicolegal status, or of using it as a basis for compensation

*Second Stage* This most typical stage has been characterized by the distinctive distribution of small, rounded densities, varying in size from a pinhead to a pea, throughout both lungs (Figure 11) The nodules appear first on

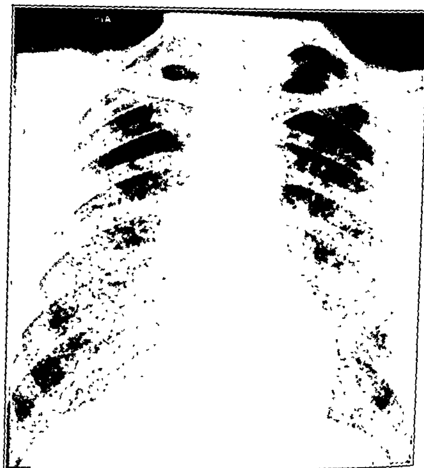


FIGURE 11

Simple silicosis showing the uniform distribution of small, round densities throughout both lungs, in a hard coal miner.

the right side, around the root of the lung, and usually become quite perceptible here before they are seen on the left side This predominance is apparent until the distribution becomes quite general throughout both lungs, and then there



FIGURE 12

Silicosis with infection and beginning coalescence of the nodules in both lung fields especially in both upper lobes in a hard coal miner with an active tuberculous infection There is a small pneumothorax in the right side

is no appreciable difference The spots are always more numerous around the hila and less in the basilar portion of the lungs The densest and most sharply defined nodules are usually found in association with the more rapidly fibrosing

dusts, such as those to which many abrasive tool grinders are exposed. There is always more or less evidence of diffuse fibrosis present, as a continuation of that often visibly started in the first stage, and as a forerunner of the essential process of the third It may not be apparent because of emphysema. When not apparent, its presence may be suggested by lack of lung expansion and diaphragmatic excursion, or sharp peaking of the domes. Theoretically, one would expect this second stage nodulation to be superimposed upon first stage appearances, but very often, the latter are not apparent. They may be obscured by emphysema, but this is not the sole cause.

*Third Stage.* This stage has been characterized by manifestations due to a predominance of diffuse fibrosis, which may present three fairly definite and distinct appearances, although all of them may be observed in the same case:

(1) The larger nodules of the second stage, where most prevalent, either may coalesce into very much larger and irregular masses, or be found close together with more or less haze between them. It may be difficult in such cases to differentiate between second and third stages, except by a lack of expansion and diaphragmatic excursion, and the presence or absence of diaphragmatic irregularities. (Figure 12)

(2) A more or less diffuse fibrosis, somewhat similar in appearance to the fibrotic process representing the late result of an extensive bilateral chronic pulmonary tuberculosis. Usually, definite nodules are still present, although not always

(3) The presence of massive fibrotic areas presenting the appearance of extensive pulmonary consolidations. In the late period of the stage, there is usually one such area on each side, rather symmetrically located subapically, but sometimes unilateral, or more extensive on one side than the other. (Figures 13 and 14) In an earlier period of the stage,



FIGURE 13

Anthracosilicosis with infection Some of the lesions are healed Other lesions, such as the one in the right lower lobe, extend to the periphery of the anterior chest wall Note that the massive lesions in the upper lung fields have a clear zone of lung around them Another interesting feature is the absence of any evidence of nodulation



FIGURE 14

Close-up of a massive or conglomerate lesion in the right upper lobe The lesion is centrally placed and does not extend to the periphery at any point From the roentgen standpoint, this lesion is regarded as silicosis with a quiescent infection.



FIGURE 15

Lateral view of the chest of an individual having anthracosilicosis Note that the major portion of the lesion is in the upper half of the lower lobe and the lower half of the upper lobe

when small, they may appear quite asymmetrical Lateral roentgenograms have shown us that many or most of thes



FIGURE 16
Massive lesions in a hard coal miner Note the symmetrical position of the masses

subapical masses are in the apices of the lower lobes, and numerous autopsy reports in the literature have confirmed this location (Figure 15) Often, associated with these

manifestations, dense, fibrous bands are found extending in various directions, but prominently downward, and frequently causing marked diaphragmatic deformities. They often resemble thickened trunks The heart and vessels are quite likely to be displaced when these bands are present Diaphragmatic excursion is very much restricted or entirely absent (Figure 16)

*Other Classifications* The present general classification of South Africa is as follows.

1 Rather more fibrosis than usual
2. More fibrosis than usual
3 More fibrosis full

| | |
|---|---|
| 4. More fibrosis to commencing fibrosis<br>5. Commencing fibrosis<br>6 Commencing fibrosis plus | Ante primary |
| 7 Commencing to early<br>8 Early commencing<br>9 Early to medium | Primary |
| 10 Medium fibrosis<br>11. Medium to advanced fibrosis<br>12. Advanced fibrosis | Secondary |

Complicated classifications are more apt to become popular in the examinations of individuals in a single industry than when several industries are studied, as seems to have been the case in connection with the South African gold miners, and as was the case with Jarvis[41, 42, 43] in his studies of the Barre granite cutters. They are, no doubt, admirably adapted for use when practically the entire work of an in-

dividual or board is restricted to the examination of silicotic persons, especially in one industry, but they are hardly practicable for roentgenologists in general, who are more favorable to simple classifications.

In the formulation of the most excellent Workmen's Compensation Act of Ontario, especially in respect to silicosis, the clinical classification of the South African Board has been embodied. This section of the Ontario Act reads as follows

(9) — (a) 'Silicosis' shall mean silicosis of the lungs (a fibroid condition of the lungs caused by the inhalation of silica dust)

(b) A person shall for the purpose of this Act be deemed to have or to have had silicosis, —

(I) In the ante-primary stage when it is found by the Board that the earliest detectable specific physical signs of silicosis are or have been present, whether or not capacity for work is or has been impaired by such silicosis;

(II) In the primary stage, when it is found by the Board that definite and specific physical signs of silicosis are or have been present, and that capacity for work is or has been impaired by that disease, though not seriously and permanently,

(III) In the secondary stage, when it is found by the Board that definite and specific physical signs of silicosis are or have been present, and that capacity for work is or has been seriously and permanently impaired by that disease, or when it is found by the Board that tuberculosis with silicosis is or has been present.

The 1925 South African law defines three legal stages of silicosis practically identical with the above Ontario specifications. In view of the greatly altered aspect of the disease in South Africa, it has become necessary to recognize addi-

tional *clinical* groups of cases, which Watkins-Pitchford[44] summarizes somewhat as follows

(a) *Simple Silicosis* The condition in an individual who remains free of overt tuberculosis It is non-progressive and produces but little effect upon the health From the pathological standpoint, these subjects present numerous small, inert nodules of dense fibrous tissue, symmetrically distributed Each nodule is sharply defined from the surrounding tissue, and is so well encapsulated as to be easily shelled out They vary in size from 3 to 4 millimeters and are rarely over 6 millimeters If closely aggregated, they may produce a certain amount of disability, mainly shortness of breath The roentgenological examination is more reliable than the clinical one

(b) *Tuberculosis with Silicosis.* This is a progressive and serious disease associated with larger nodules unsymmetrically distributed and composed of young connective tissue elements among which tubercle bacilli are found. Adjacent nodules are frequently aggregated into masses There are 2 classes of cases in this group (1) The individual may have had simple silicosis at first, followed by the development of unsymmetrical tuberculous lesions (*tuberculosilicosis*) This change may occur after many years (2) The tuberculous lesions may be manifest first, followed by the silicotic ones in close association (*silicotuberculosis*) Here again, the roentgenographic findings are important In tuberculo silicosis, the hitherto uniformly mottled areas are now invaded by one or more unsymmetrical areas of shadow with mottling around them The Miners' Phthisis Act now provides separately for these two general groups of cases, as has been the case in Ontario.

At the meeting of the First International Conference on Silicosis[45] at Johannesburg, in August, 1930, the three clinical stages recognized locally were accepted by the Conference.

They were as follows: *First Stage* — Respiratory symptoms slight, few or no physical signs and capacity for work little impaired. Roentgenograms show the linear shadows increased and present discrete shadows of nodulation. *Second Stage* — All physical signs are increased. The nodular shadows are increased in number and size and show a tendency toward confluence. *Third Stage* — All signs and symptoms are greatly accentuated, and there is a total loss of working capacity. When tuberculosis is present, the stage classification must be based more or less upon a loss of working capacity than upon physical signs and roentgenographic appearances. It was recommended that an internationally comparable roentgenographic technic and terminology be adopted, and that a further study be made of the correlation of roentgen appearances, pathology and symptomatology of silicosis with or without tuberculosis

In 1931, Dr Pancoast and I decided that it was more rational to dispense with any attempts to employ general stages of progression in any of the ways so far discussed and to substitute a pathologic-roentgenological classification, which could be combined with the clinical aspect peculiar to any one industry, or with all of them. Those who have studied a single phase of pneumoconiosis in one occupational group, such as asbestos workers, coal and hard rock miners, granite cutters, and sand pulverizers, for example, have found it most difficult to apply any definite numerical order of progression by stages such as that used for other occupations. Likewise, those who study a large number of occupations and industries find it difficult to adhere to a general division into stages, such as that, for example, as had its origin in the study of hard rock miners, without making exceptions or creating certain variants. This becomes more complicated with each new occupation studied. Pneumoconiosis implies a condition of fibrosis, occurring, so far as we

know, in only one general way, but with certain variations dependent upon uncertain or unknown influences.

Now, the clinical phenomena must, of necessity, depend upon obvious forms of pathological changes Furthermore, roentgenographic appearances portray these pathological changes as soon as they become macroscopic, or reveal their influences while the actual changes are still microscopic Therefore, it seemed wiser to substitute, for a numerical progression of pathological changes typical for a few of the occupations, a general pathological classification which would include the typical and the variants, and, at the same time, be applicable to both clinical and roentgenological studies for each industry or occupation.

A study of the diagrammatic representation of the lymphatic system of the lungs, such as that of Miller,[46, 47] and an application of the pathological features of the condition to this serves to indicate that there are practically three simultaneous primary distributions or accumulation points for dust, which correspond to later possible manifestations of fibrosis (Figure 7) These have been summarized as follows: (1) Blockage of the peribronchial and perivascular lymphatic systems, with later intensification of trunk shadows, linear markings, and pulmonary lymph nodes, (2) Lodgment in lymphoid deposits, with subsequent development of nodular fibrosis, (3) Thoroughly interstitial fibrosis following a rapid block of lymphatic channels To these are added the pleural side-track, which is more or less common to all cases and needs no special classification

From a study of various industries, it seemed evident that fibrosis might exhibit *predominance* in any of these distributions in individual occupations For example, in coal mining, the driller may show predominance of the nodular type, whereas the man in the breaker may exhibit only the first stage for many years, in hard rock mining, the nodular type

predominates until diffuse fibrosis supervenes ; in the granite cutter, the interstitial type predominates, although the nodular may, exceptionally, do so ; in the asbestos worker, the interstitial type is characteristically predominant. Interstitial predominance, rapidly progressing, will develop into diffuse fibrosis without nodular predominance. And so we might run through all the fibrosis producing dusty occupations.

The following pathologic-roentgenological classification was offered as a suggestion to overcome the existing confusion .

| | |
|---|---|
| 1. Peribronchial-perivascular-lymph node predominance | rapid<br>slow |
| 2 Early interstitial predominance ( interferes with diaphragmatic movement ) | with nodular appearance<br>without nodular appearance<br>rapid or slow |
| 3 Late or advanced interstitial predominance | |
| 4 Nodular predominance | non-progressive<br>progressive |
| 5 Advanced diffuse or terminal fibrosis | conglomerate nodular type<br>interstitial type<br>massive fibrosis type |

It was thought that many individuals might show, or occupations be characterized by, various combinations of these types ; and that, in one industry or occupation, there would be a sequence of predominant appearances which would correspond closely to the clinical aspect and progression of

the disease. This has not been true in my experience, however, even in studies of individuals exposed to pure silica without contaminating dusts.

We found it difficult to understand, or to explain, all of the underlying factors in the rapidly developing interstitial fibrosis Gardner,[48] however, offered an explanation He stated that the obstruction in the deep pulmonary lymphatics diverts the lymph flow and dust cells toward the pleura, where counter-resistance is met Consequently, dust cells pass through the walls of the lymph vessels and accumulate in the surrounding connective tissue at a point where the two lymphatic systems communicate In support of the theory first advanced,[40] of the choking of lymphatic channels by dust cells and the contraction of their lumen, which has been open to criticism, Gardner believed that the point of actual obstruction could be in the pulmonary lymph nodes, which would produce stasis in both deep and superficial lymphatics He further suggested that the previous condition of these lymph nodes, as to changes induced by childhood tuberculosis and other agencies, might have some influence on the rapidity of onset and progression of silicosis It is my feeling that active infection may exert an important influence upon the roentgen manifestations.

These roentgenological phases of the condition, which we have described previously,[49 50 101] may be briefly summarized as follows

1. The perivascular-peribronchial-lymph node aspect due to the relaying of phagocyted dust to the pulmonary lymph nodes and their subsequent enlargement and ultimate partial fibrosis to the gradual enlargement of lymphoid deposits along the course of lymph vessels and the subsequent thickening of these vessels and stasis of contents This is characterized roentgenographically by increased prominence of the hilum and trunk shadows and linear markings This appear-

ance is by no means characteristic of pneumoconiosis alone, and even if it does indicate the condition, the phase is absolutely not incapacitating It, together with a barely perceptible appearance of macroscopic nodules, corresponds to the so-called first stage.

2. The nodular aspect is due to the gradual enlargement of lymphoid deposits and their coalescence into quite apparent macroscopic nodules symmetrically scattered throughout both lungs. This corresponds to the so-called second stage. It is conspicuously absent in many instances, especially when the silica intake is rapid.

3 The interstitial type of the condition results from a hilumward and pleuralward block in the lymphatics and the escape of dust phagocytes in large numbers into the interstitial interalveolar tissue and subsequent fibrosis It appears as a faint homogeneous haze, first on the right side, then on the left If the silica intake is comparatively slow, it may accompany the perivascular-peribronchial-lymph node aspect, but if more rapid, it may be associated with the nodular type, or may progress without the latter directly into the terminal stage of the condition without any evidence of nodulation The unprotected or inadequately protected sand blaster, sand pulverizer, and sandstone abrasive worker have been among those especially prone to present this rapid interstitial aspect

4 Terminal and incapacitating silicosis is characterized by two general appearances, — a terminal diffuse fibrosis of a conglomerate nodular type, one which is quite similar in appearance to a generalized chronic fibroid tuberculosis, and the terminal stage characterized by large consolidated areas

In 1935, another classification or silicosis terminology was suggested because it was felt that, if objective terms, descriptive of the type of pathological change, could be generally adopted, material progress would result. The committee's

report,[51] given below, will certainly be revised from time to time as experience directs. Abstracts from the report are as follows:

It should be distinctly understood that the tabulation which follows applies only to *silicosis*, that form of pneumoconiosis resulting from the inhalation of dust with a high silica ( $SiO_2$ ) content.* Other forms, like asbestosis, are excluded from this consideration because their pathology is essentially different from that of silicosis.

The tabulation contains two columns, on the left, the roentgenological appearances; and on the right, the corresponding pathological lesions. There is further subdivision to describe the appearance of, ( 1 ) the healthy lung, ( 2 ) the uncomplicated silicotic lung, and ( 3 ) the lung of silicosis with infection. The changes described under the first division are those compatible with a state of good health; and, *while they may be produced by the inhalation of relatively* small amounts of silica dust, they are not sufficiently characteristic or advanced to substantiate a diagnosis of silicosis. Similar or identical appearances may also result from the inhalation or nonsiliceous dusts, from certain infections, from cardio-vascular disease, and from certain other rare conditions. The changes involved are, for the most part, confined to the lymphatics and perilymphatic connective tissues and do not affect the parenchyma of the lung. Since, by definition, silicosis is a disease characterized by nodular fibrosis in the parenchyma of the lung, these alterations, even when they may have been caused by inhaled silica, do not constitute a basis for a diagnosis of silicosis. The second group covers the discrete and conglomerate nodular fibrotic reactions of sim-

* Some of the nonsiliceous components of certain industrial dusts seem to modify the pathological reaction, but the character of shadows cast by these modified lesions is not sufficiently defined at the present time to include them in the tabulation. Later, when more information has accumulated, certain other terms may have to be included.

ple silicosis. The last group deals with silicosis complicated by infection In the majority of instances, the *infecting organism is* the tubercle bacillus, but the classification is sufficiently broad to include other types of infection. *Certain criteria* by which one attempts to differentiate various forms of infection will be discussed

| *Roentgenological Appearances* | *Histological Appearances* |
|---|---|
| HEALTHY LUNGS AND ADNEXA | |
| 1 *Healthy lungs* — As defined by the N.T.A. Committee report | 1. Essentially the normal tissues of the vascular tree, the mediastinum, the bronchi, and trachea |
| 2 Irregular exaggeration of the linear markings, with possibly some beading confined to the trunks | 2. Cellular connective tissue proliferation about lymphatic trunks in the walls of vessels and bronchi Beading may be due to various causes, as blood vessels seen end on, arteriosclerosis, minute areas of fibrosis in lymphoid tissues along the trunks. |
| 3 Increased root shadow | 3 Cellular reaction in the tracheo bronchial lymph nodes with extensions along afferent lymphatic trunks |

These changes come within normal variations when not accompanied by recognized organic disease. *Irregular exaggeration of the linear markings, with possibly some beading,* belongs in the healthy chest group even when found in persons with a history of considerable exposure to silica, for such changes are nonspecific in character and they do not involve the parenchyma of the lung. Silicosis as a clinical

disease begins only when the lung proper is affected. Likewise, under 3, *Increased root shadow* may be of nonspecific origin and hence is not diagnostic. In the *early* stages of silicosis, the mediastinal shadow may be widened, owing to the enlargement of the tracheobronchial lymph nodes from accumulated dust and cellular reaction to it; later, when specific fibrosis develops, the tissues generally contract and the nodes decrease in size. The changes described under 2 and 3 may be caused by many forms of irritation; if they are due to silica, they are identifiable only by microscopic examination. They do not, apparently, interfere with respiratory function, and they are not of diagnostic significance.

| *Roentgenological Appearances* | *Histological Appearances* |
|---|---|
| SIMPLE SILICOSIS | |
| 4 *Nodulation* — Discrete shadows not exceeding 6 mm in diameter, tending to uniformity in size, density, and bilateral distribution, with well defined borders surrounded by apparently normal lung shadow. The outer and lower lung fields characteristically show fewer nodules. | 4 Circumscribed nodules of hyaline fibrous located in the parenchyma of the lung. Occasionally, some of these nodules may show microscopic foci of central necrosis. |
| 5 Conglomerate shadows that appear to result from a combination or consolidation of nodulation usually with associated emphysema manifested by —<br>*a* Localized increased transparency of the lung with loss of fine detail | 5 The result of coalescence of discrete nodules; an area in which the nodules are closely packed and most of the intervening lung is replaced by more or less hyaline fibrous tissue. The lung architecture is partially obscured. No demon- |

| *Roentgenological Appearances* | *Histological Appearances* |
|---|---|
| SIMPLE SILICOSIS — continued | |
| *b* Intensification of the trunk shadows by contrast<br>*c* Depression of the domes with possible tendency toward individualization of the costal components of the diaphragm.<br>*d* Lateral view: Increase in the preaortic and retrocardiac space with exaggerated backward bowing of the spine. Widening of the spaces between the ribs may or may not be present. | strable evidence of infection. Emphysema is a compensatory dilatation of the air spaces with or without thickening of the septa |

The second group of changes is limited to *simple silicosis* uncomplicated by demonstrable signs of infection This condition is characterized by the presence of small, discrete nodules of fibrous tissue disseminated throughout the functional parts of both lungs. The lesions and the shadows cast by them tend to be spherical, hard, sharply defined, and vary in size from 2 to 6 mm While the distribution is usually uniform throughout both lungs, the extreme apices and the outer portion of the bases are frequently uninvolved In less advanced cases, the nodules remain discrete and separated by air-containing tissue.

Number 5 deals with the *conglomerate shadows* of simple silicosis, which appear to develop from a combination or consolidation of discrete nodules. The resultant lesion and the shadow that it casts are often difficult to distinguish from the *massive shadows* of silicosis with infection, 9 It is gen-

erally assumed that conglomeration results from accidental overlapping and fusion of discrete nodules when they become very numerous, but since conglomeration is usually a localized affair and does not occur in the same position of the lung of every individual, it is logical to enquire why the nodules happen to fuse in one portion of the lung and not in others Microscopic examination of the tissues from such areas reveals no evidence of active infection The nodules *seem to be much closer together than in other portions of* the lung, they are less uniform in size, and they are usually embedded in a matrix of diffuse fibrous tissue having the same characteristic hyaline appearance as that forming the nodules themselves It seems probable that conglomeration may have occurred because the portion of the lung in question was previously damaged by a localized inflammatory process occurring before or during the early period of dust exposure Because the tissue was injured, more dust would tend to accumulate in the area, the nodules would develop irregularly and would frequently be very close together The silica lodging in pre-existing granulation or scar tissue would exert its characteristic effect, and a diffuse hyalinization would result This explanation for conglomerate reaction is, at present, hypothetical, proof will come from long continued serial roentgenographic studies of groups of persons exposed to silica dust and from the chance autopsy that may be obtainable To differentiate *conglomerate shadows* from the *massive shadows* of infection, 9, reliance must be placed upon the absence of change in size and character of the shadows in serial films taken over an extended period of time and upon the clinical findings in the case

Emphysema is usually associated with far advanced silicosis and it is particularly liable to complicate conglomerate nodulation It occurs in the immediate vicinity of the conglomeration as a result of the distortion produced by con-

tracting scar tissue; there is, also, a generalized 'compensatory' emphysema found along the borders of the lung, particularly at the bases. The latter type is also common in far-advanced generalized nodulation.

AUTHOR'S NOTE: I am not at all certain that conglomerate shadows and even nodulation should be included under a classification of simple or uncomplicated silicosis. My experience in an industry of pure silica workers rather points to some complicating factor. I am almost convinced that shadows, such as described above, can be explained by a complicating healed infection. This will be discussed later.

| *Roentgenological Appearances* | *Histological Appearances* |
|---|---|

SILICOSIS WITH INFECTION

The characteristic appearances described under simple silicosis are modified by infection as follows:

| | |
|---|---|
| 6. Localized discrete densities and/or string-like shadows accompanying those of simple silicosis described above. | 6. Strands of fibrous tissue, often along trunks and septa, with or without areas of calcification; indicative of 'healed' infection. |
| 7. *Mottling* — Shadows varying in size with ill-defined borders and lacking uniformity in density and distribution, accompanying simple silicosis. | 7. (*a*) Areas of bronchopneumonia with or without caseation, i.e., acute infection.<br>(*b*) Lobular areas of proliferative reaction with or without caseation, i.e., chronic infection. |
| 8. *Soft nodulation* — The nodular shadows described under simple silicosis, 4, have now assumed fuzzy borders and/or | 8. Perinodular cellular reaction either exudative or proliferative in character. |

| *Roentgenological Appearances* | *Histological Appearances* |
|---|---|
| SILICOSIS WITH INFECTION — *continued* | |
| irregularities in distribution This change may or may not accompany the simple mottling of 7 | |
| 9 *Massive shadows* of homogeneous density not of pleural origin symmetrically or asymmetrically distributed | 9 Extensive areas of fibrosis probably due to organized pneumonia of tuberculous or nontuberculous origin superimposed upon a coexisting silicotic process Outlines of normal structures may be partially destroyed |

In the last group, *silicosis with infection*, are included all cases with detectable evidence of infection whether active or inactive. In this respect, we depart from the South African procedure, which includes here only active infection The difficulty of determining activity, particularly in the silicotic subject, is our chief reason for this arrangement

Number 6 covers foci of healed infection Identification of such changes depends upon the same criteria that are generally employed in otherwise normal individuals In the silicotic subject, the shadows usually occur upon a background of generalized nodulation, although in some cases, there may be a distinct tendency toward excessive nodulation in the immediate vicinity of the scars left by the infection Where the exposure to dust has been limited, the major evidence of nodulation may occur about the foci of healed infection with much less reaction in the remainder of the lung The string-like shadows of healed fibroid tuberculosis

are not difficult to interpret if they occur in the classical location, i e, in the upper third of the lung. In the lower lung, they present a problem whose solution depends largely upon the experience of the roentgenologist.

The term, *mottling*, 7, we have reserved to describe the shadows of infectious lesions in contradistinction to *nodulation*, which is restricted to those of the silicotic dust nodule. It is essential that this distinction be appreciated and recorded in the terminology. In tuberculosis, mottling is due to bronchogenic or aspiration foci of disease which exhibit a characteristic clustered arrangement. The lesions may be exudative (acute) or productive (chronic) in type; the difference will be registered in the roentgenogram by a mottling which is fluffy and ill-defined, or hard and sharply defined, as the case may be The distribution of the mottled foci, together with the presence of large foci of older disease interpreted as tuberculosis, and clinical and laboratory findings establish the character of the infection. Mottling due to chronic infection that has developed previous to or simultaneous with the relatively early periods of dust exposure may exhibit little or no effect from the inhaled silica for many years In nontuberculous broncho-pneumonias, the large chronic foci are absent, and the disseminate mottling may involve different parts of one lung or of both lungs In many instances, the nature of the infection must be established by serial examinations over considerable periods of time and by careful correlation with clinical and bacteriological findings.

*Soft nodulation*, 8, is a term that has been coined to describe a rather uncommon combination of silicosis with infection, usually tuberculous The ordinary hard, sharply defined nodular shadows of simple silicosis, under these circumstances, appear to have enlarged and lost definition Their borders are now fuzzy and blend imperceptibly with the sur-

rounding lung structure. Such lesions generally occur in association with localized conglomerate shadows in the apex or other portions of the lung. Histologically, the infection appears to have localized in and about pre-existing silicotic nodules, so that each is surrounded by a zone of exudative or productive cellular reaction.

*Massive shadows of homogeneous density*, 9, are cast by the areas of combined silicosis and infection, usually chronic in nature. The two processes appear to have developed simultaneously and unusual amounts of dust accumulate in the diseased area. Generalized nodulation usually occurs throughout the remainder of the lungs. Pleural densities can be differentiated in stereoroentgenograms, and by overexposure, it often becomes possible to penetrate the extremely dense intrapulmonary areas and analyze their internal structure. Not infrequently, cavities may be visualized that were completely overlooked with the usual technic. When due to tuberculosis, such lesions are often bilaterally symmetrical. If the process extends to the pleural surface, a tuberculous etiology is postulated, while other infections are more often deep seated.

Histological examination of such lesions shows conglomerations of simple nodules embedded in masses of more or less perfectly organized granulation tissue. Often the fibrous tissue has undergone the same peculiar hyalinization that characterizes the interior of the silicotic nodule. Usually, the outlines of the original lung architecture are completely destroyed. Manifestations of infection depend upon the nature of the process. If tuberculous, there will be foci of caseation and possibly small cavities. Calcification is not infrequent. If the process is inactive, the presence of fibrous tubercles, which do not exhibit the hyalinization of silicosis, may be present. The occurrence of giant cells is helpful. A partially organized nontuberculous pneumonia usually contains foci

distributed throughout the lung, more on the right at first There is a slight homogeneous haze, beginning in mid-lung fields At this time, there is no interference in diaphragmatic movement. There is, occasionally, thickening of the interlobar pleura (Figure 11)

The above observations are included under simple silicosis only because of the fact that those most interested in this subject support such statements. I have always accepted early nodulation as evidence of simple silicosis, until it was possible to study a group of workers in a pure silica industry in which some of the workers were exposed to large quantities of finely divided silica over a period of years. In some instances, the exposure period was over forty years. In spite of such exposures, a large percentage of the workers showed no changes in their lungs. Individual lack of susceptibility has not seemed an adequate explanation to me for such observations.

*Differential Diagnosis.* — There are many conditions that will cause lung changes which simulate the roentgen findings of silicosis Some of these affections have been definitely identified, others have not.

*Metastatic Malignant Conditions of the Lungs* — Most cases of this kind are readily differentiated from nodular silicosis, but occasionally, one encounters a case in which appearances are very similar, because the metastatic nodules are large and widely separated. (Figure 17)

*Tuberculosis* — These nodules are small, soft and fluffy, unlike the smaller, dense ones in silicosis, yet in some cases, it is impossible, either with clinical or roentgenographic evidence to differentiate the two conditions. The history will greatly aid in reaching a definite conclusion Roentgenographically, the main variation may be the difference in size and density of the hila, although this is not always the same (Figure 18)

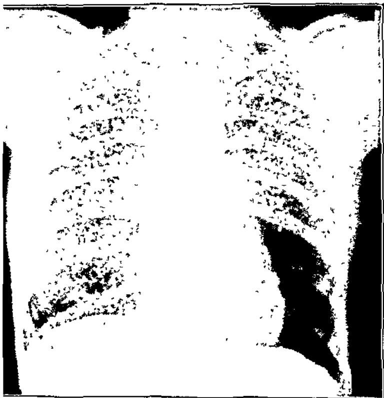


*Figure 18*

Miliary tuberculosis This appearance could readily be mistaken for that of a nodular type of silicosis

*Mycotic Infections* —Actinomycosis, sporotrichosis, leptothrix, and other fungoid infestations may cause nodulation in the lung and simulate silicosis Such nodules, however, are usually larger, softer, and of considerable variation in size *In proven mycotic infections, dyspnea has been an outstand-*

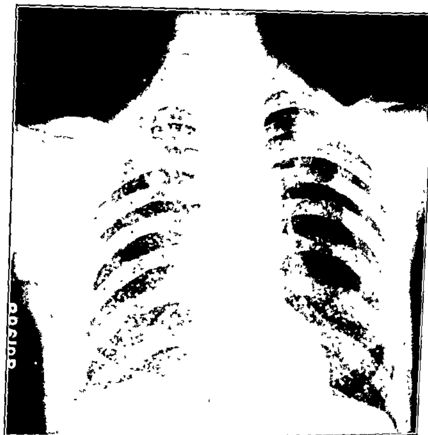


FIGURE 19

Miliary calcification of unknown origin This patient had a carcinoma of the stomach and the miliary calcification in the lungs was a coincidental finding He had never been exposed to dust and had never worked in the wheat fields Dr. LeRoy U Gardner studied pathological sections obtained at post-mortem examination and was unable to determine the cause of the calcification

ing symptom. Fawcitt [52] believes that these conditions are more prevalent than is commonly thought

*Miliary Calcifications* — Most roentgenologists have had

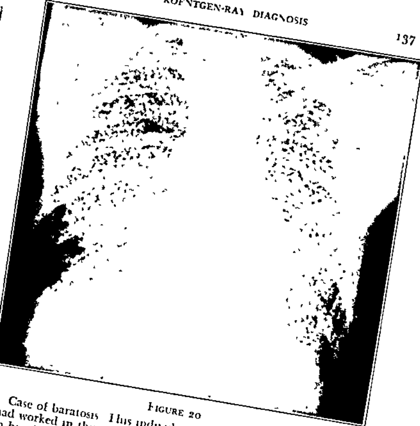


FIGURE 20

Case of baratosis This individual worked in barium rock and
had worked in this industry for a number of years The findings
in his chest roentgenograms were entirely coincidental and were
not causing any symptoms The individual was not incapacitated,
in fact he had few, if any, symptoms A study of the roentgeno-
grams made approximately seven years before the one illustrated
above showed a fine, discrete nodulation at that time, but not
nearly so marked as is evident at present

experience of seeing one or more cases of multiple sym-
metrical lung calcifications, and most of us have regarded
them as evidences of healed miliary tuberculosis, with or

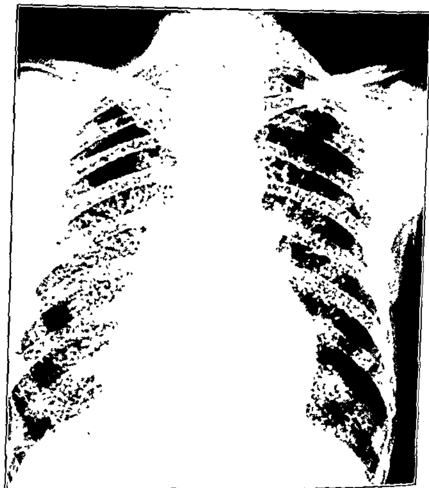


FIGURE 21

Soft nodulation in a hard coal miner, diagnosed as silicosis with infection Note the tendency toward coalescence in the middle third of the right lung

without good reason to so diagnose the condition The appearance is not likely to be confused with silicosis Sayers and Meriwether had the unusual experience of finding 125

such cases in an examination of 18,285 men at the Pilcher, Oklahoma, Mining Clinic Many, but not all, of these men had been miners, and some of those, who had been, showed real evidences of pneumoconiosis in addition (Figure 19) The lungs showed a diffuse distribution of dense, discrete, apparently calcified, shot-like spots throughout the lungs They were of all sizes up to 1 cm in diameter, and were variously ascribed to fungus infections, pneumomycosis, and healed miliary tuberculosis

*Baritosis* — I have seen several patients with widespread dense nodulation, typical of that seen in so-called simple silicosis, and similar to the cases seen in Italy [51] These individuals have been exposed to barium dust, which may be responsible for the changes observed in their lungs. (Figure 20)

*Silicosis with Infection* (nodular predominance type) — Pulmonary tuberculosis is a frequent complication of pneumoconiosis. The latter condition may be a predisposing factor in the frequent incidence and serious consequences of the infection, especially when the progress of the pneumoconiosis is rapid The two conditions often must be differentiated when only one exists, or pneumoconiosis is present in only a minor degree and presumably not sufficient to exert any predisposition Differentiation is necessary because the two conditions may produce quite similar roentgenologic appearances (Figure 21)

Tuberculous infection may be present in what appears to be a purely nodular type of silicosis (Figure 22) It is far more likely to be a superimposed process, and presumably exogenous in the absence of any typical adult lesion When the nodules tend to become conglomerate, or there is the appearance of considerable interstitial change between them, we may suspect a superimposed tuberculosis, although it can not be absolutely proven In the typical nodular type, the shadows vary in size from those barely visible to that of a

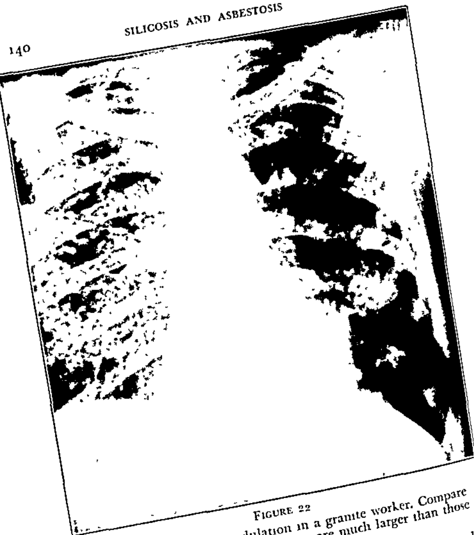


FIGURE 22

Perinodular or soft nodulation in a granite worker. Compare with Fig 21. The nodules in Fig 22 are much larger than those seen in Fig 21

small pea; they are more or less regular in outline and rather sharply circumscribed. The density depends considerably upon the size The question of superimposed tuberculous infection may also arise when the nodular shadows




FIGURE 24A

A rather typical lesion seen in an individual having silicosis with a healed infection. These lesions are often bilateral and often found in the upper third of the lung fields Note the aerated lung surrounding these areas

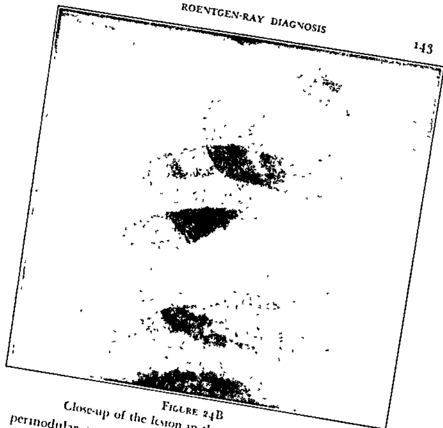


FIGURE 24B
Close-up of the lesion in the right upper lobe

perinodular type (Figure 22) especially if this is occurring in some nodules and not in others. To make this distinction by a single examination is likely to be guess work. Even serial observations must be made by the use of exactly identical technic.

It is entirely possible that other infections may complicate an already established silicotic process in the lungs, and, in the absence of other clinical or laboratory observations, it may be impossible to identify the nature of the contaminating

influence Unless the infection be active, as indicated by changing roentgen appearances, determined by serial roentgenograms or by clinical observations, one may justifiably regard the lesion as silicosis with healed infection. (Figure 23)

*Silicosis with Healed Infection* (*conglomerate nodular type*) — The roentgen findings are as follows: The hila may be increased in prominence, but usually are not noticeable The trunk and other lung markings may be increased There are conglomerate masses and beginning massive fibrotic masses which do not extend to the periphery of the lung and are usually found in the upper two-thirds of the lung fields, involving either the upper lobe bases or apices of the lower lobe. (Figure 24) The lower third of the lung is emphysematous, as a rule. The trachea is not displaced, unless there has been a pre-existing tuberculous process. There is moderate interference with diaphragmatic movement, with possible peaking, humping or waviness of the domes. Not infrequently, there is thickening of the interlobar pleura.

In previous communications on this subject, I have regarded the above findings as indicative of a simple silicotic process Increasing experience with pure silica workers has lead me to believe that such roentgen appearances represent silicosis with a healed infection, or at least a quiescent period in the infective process During the active period of the infection, the lesion extends to the periphery of the lung As the infection subsides, the conglomerate mass is found more mesially, with a clear lung area peripheral to it Such observations as these have taught me to feel more optimistic as to the future in the treatment of silicosis with active infection. (Figure 25)

I have considered, under the healthy chest, a possible explanation for the location of the conglomerate lesions. It is in such lesions as these that the massage action of the thoracic cage, heart, and diaphragm upon the lymphatic flow may be

of major importance With the subsidence of the active infection and its accompanying fixation, the lung structures can again begin to move relatively freely. In my experience, it has seemed that the extent of the lesion has progressed more readily in those areas of the lung (upper lung fields) that are more susceptible to fixation or interference with respiratory movements. (Figures 9 and 25)

*Silicosis with Active Infection* (conglomerate nodular type) — Pulmonary tuberculosis may be present before a silicotic process becomes predominant, or it may occur afterward. If the infection is active, the lesion extends to the periphery, usually *It should be borne in mind that oblique or lateral views may be essential to diagnose or exclude peripheral extension of a lesion situated in certain portions of the lung*

If the silicotic process was present before the tuberculous infection, the trachea usually remains in the midline, whereas, if the tuberculous process was present first, the trachea is more likely to be displaced, either to the side or anteriorly or posteriorly Usually, pulmonary tuberculosis, with the possible exception of Friedlander's pneumonia, can, for practical purposes, be regarded as the cause of tracheal displacement

In the larger lesions, heavily exposed roentgenograms, or those made with the aid of the Potter-Bucky diaphragm, may assist in demonstrating a cavity or calcium nodules, as a means of identifying the nature of the infective process.

I have seen only a few individuals in whom silicosis with active infection was complicated by a spontaneous pneumothorax All of these patients did poorly. One wonders, therefore, if such observations may not be regarded as collateral evidence against the use of collapse therapy in the treatment of silico tuberculosis

*Differential Diagnosis.*[100] — *Infiltrating or Permeating Malignant Metastases* — Quite frequently, metastatic malignant

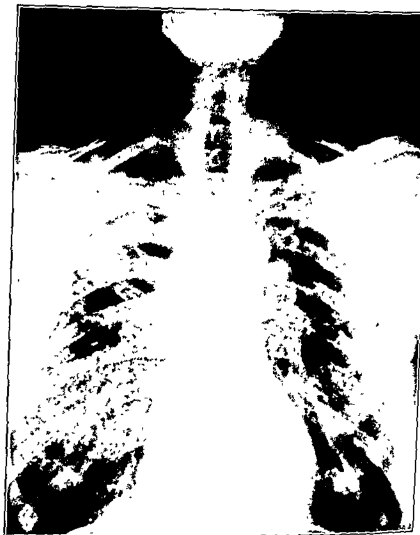


FIGURE 25A

Silicosis with infection There are a few small, discrete nodules distributed throughout both lung fields In the right upper lung field, there is a massive lesion which extends to the periphery



FIGURE 25B

Five years later. The lesion in the right upper lung field has
ontracted down to a relatively much smaller mass, and there is
w aerated lung around it. Elsewhere, the nodules in the lungs
ve not changed. This individual is working in pure silica dust,
uch contained high concentrations of finely divided silica up
il a few years ago. This individual at one time, had a silicosis
h active infection. He now has silicosis with an inactive infec-

processes in the lungs are of such a character as to take on an appearance closely simulating that of pneumoconiosis of a conglomerate type Naturally, the individual is incapacitated, and often in a manner similar to a silicotic patient.

*Polycythemia or Erythremia.* — This condition may be regarded from two points of view. As an independent disease, it usually has an associated increased prominence of the hilar and trunk shadows, due to the engorged pulmonary vessels On the other hand, high red cell counts, even sufficient to suggest polycythemia, are very frequently associated with pneumoconiosis There seems to be reason for believing that lung fibrosis and pulmonary arteriosclerosis can produce a condition akin, perhaps, to polycythemia and due, presumably, to impaired interchange of oxygen and carbon dioxide. Yater and Constam [54] reported two cases of primary pulmonary arteriosclerosis, one an anthracotic patient with emphysema, and the other with a mitral stenosis They stated that secondary pulmonary arteriosclerosis involves the larger vessels and may be due to mitral stenosis, emphysema, congenital heart disease, and conditions in which the lung volume is reduced, as tuberculosis and tumor. Polycythemia is one of the clinical phenomena Rosenthal [55] reported three cases of workers in dusty atmospheres or with irritating gases in whom there developed cyanosis and dyspnea, and true polycythemia in one. Moschowitz [56] believes that secondary pulmonary arteriosclerosis is very common He found it in 6 5 per cent of autopsies and places the causes in the order of frequency as, mitral stenosis, emphysema, pulmonary fibrosis, extensive pleural adhesions, kyphoscoliosis, and congenital heart lesions We have been interested in determining any relations between pneumoconiosis and polycythemia, and, in our search for any true associations, we have been inclined to accept the views of Wood,[57] that there are really two conditions to be considered One is a compensatory mild polycythemia

from lung fibrosis, and the other, the true erythremia originating in bone marrow changes and capable of producing alterations in the roentgenographic appearances of the chest referred to at the beginning of this paragraph

*Silicosis with Infection and Massive Lesions* — Terminal and incapacitating silicosis with infection is characterized by three general appearances — a terminal diffuse condition of a conglomerate nodular type, one which is quite similar in appearance to a generalized chronic fibroid tuberculosis, and the terminal stage characterized by large consolidated areas, which quite frequently closely resemble tuberculous consolidations Some of the lesions seem to be quiescent, with the roentgen findings as follows The trachea or hilum is not displaced The hila may be prominent, but often is not noticeable The lung markings are increased, but usually obscured by emphysema The lesions are usually in the upper two-thirds of the lung fields and do not extend to the periphery The nodules are not particularly prominent There is marked emphysema of the lower one-third of the lung field As a rule, there are no cavities, if present, they may or may not be due to vascular injuries The Potter Bucky film shows trabeculation of massive areas without calcification or cavity Frequently, there is marked interference with diaphragmatic movement Peaking of the domes of the diaphragm occurs often, and sometimes, there is thickening of the interlobar pleura.

If the tuberculous infection was present before the silicosis became manifest and the infection is active, the roentgen findings are as follows The trachea and the hila may be displaced, if pneumoconiosis was not the predominant lesion originally Usually, lung markings are largely obscured by emphysema The massive lesions are usually in the upper two thirds of the lung fields and extend to the periphery Nodular formation, if present, is generally best seen in the

lower lobes. Emphysema is marked. A Potter-Bucky film shows a homogeneous density of the lesion, with occasional cavity formation At times, there are evidences of calcification. Almost invariably, there is interference with diaphragmatic movement, with peaking of the domes of the diaphragm

One frequently observes a case in which there is a rather extensive consolidated lesion in the upper chest, more marked on one side, or limited to one side. In addition to this, there are nodular lesions, usually lower down, on one or both sides This type of case represents a most difficult one for differential diagnosis. If the case is essentially one of tuberculosis, the massive lesion is most likely a rapidly progressive one and the nodular appearance may be due, in part at least, to a bronchopneumonic spread to one or both sides

The differentiation really lies between massive consolidations of the terminal stage of silicosis with a healed infection and massive tuberculous lesions which are made up of a large amount of fibrosis as a result of the accompanying pneumoconiosis Such lesions have the contracting characteristics of any large fibrosing tuberculous areas. This may be bilateral, but the chances are that one side started before the other, or progressed further and fibrosed more than the other. One must regard these lesions as of tuberculous, or partly tuberculous, origin when they are *peripherally* located The unilateral or older bilateral lesion of this nature will cause homolateral displacement of the trachea and possibly of the vessels, and because it must retract upward and outward, the hilum shadow is apt to be similarly displaced These displacements were brought to our attention in the first American paper on the roentgenological aspect of silicosis by Lanza and Childs[39] in 1917. Silicotic consolidations with infection will not displace the mediastinal structures unless the tuberculous infection preceded the silicotic process

When there are bilateral consolidations, one of which is obviously tuberculous because of the effects of retraction, the lesion on the opposite side may not extend to the periphery, and yet be tuberculous, as indicated by upward displacement of its corresponding hilum shadow. This lack of peripheral location is explained by the fact that it cannot retract upon and displace the central movable structures, like the trachea, because they have already been fixed by the contralateral lesion

There is still further differentiation to be made by roentgenological studies If the individual, consolidated lesions are intentionally over-exposed, especially with the use of the Potter-Bucky diaphragm, considerable difference will be noted in the structures The silico-tuberculous consolidation shadow will appear generally homogeneous, and any stringy appearance due to tuberculous fibrosis will be coarse and far apart, and cavities, or calcifications, or both, may be found The purely silicotic consolidation with healed infection will not be nearly so homogeneous In addition, there is apt to be a close trabeculation

## Further Considerations of Tuberculosis and Silicosis [99]

Throughout the following paragraphs and to some extent throughout the chapter, the terms 'silico-tuberculosis' and 'tuberculo-silicosis' are used These terms are rather confusing and possibly the terms should be dropped entirely

On the other hand, these terms are frequently referred to in compensation legislation and in the literature on this subject The terms 'silico-tuberculosis' and 'tuberculo-silicosis have gained a great deal of notoriety in the radiologic literature particularly and in those instances, this author at least implies one of the following ideas

a. silico-tuberculosis

1. The patient has had a well developed silicosis before tuberculosis became superimposed. In such instances, the trachea remains in the midline.

2. Silico-tuberculosis may mean that the tuberculous lesion although predominant, has had its clinical and radiologic manifestations modified to such an extent that *the roentgen and clinical signs are not those of tuberculosis* alone, even though the tuberculous process is out of proportion to that of silicosis.

b. tuberculo-silicosis

1 The term tuberculo-silicosis is employed in those instances in which tuberculosis was the primary lesion and silicosis was superimposed. In these individuals, one does not refer to childhood tuberculosis, but only to the adult type and in such instances, the trachea is usually displaced toward the tuberculous process.

It has seemed advantageous to discuss the relations of tuberculosis and silicosis under certain definite groups of cases, which will be dealt with in numerical order as follows:

1· The first group to be discussed comprises those cases of pulmonary tuberculosis in which there can be found no roentgenological evidences of silicosis after several years of service in a dust-laden atmosphere, and the condition present is obviously only tuberculosis We must bear in mind that dust exposure does not preclude the possibility of anyone contracting tuberculosis. A consideration of this group must include the differentiation of the evidences of the infection from all of the phases of pneumoconiosis. We have seen all phases of silicosis interpreted from roentgenograms when tuberculosis was obviously the only condition present. In some of these cases, compensation had been granted and in others, claimed on the basis of silicosis simply through lack of diagnostic experience, and because the claimants worked

where the production of a silica-laden atmosphere was assumed as a possibility. The differential diagnosis in this group resolves itself into the ability to recognize evidences of any of the above-mentioned phases of silicosis, on the one hand, and those of pulmonary tuberculosis on the other. There seem to be many roentgenologists who are not able to do this, although they make the attempt, and especially in connection with compensation cases. A further discussion of this group would entail a prolonged dissertation on both diseases. It should be borne in mind that this group does not include cases which may have both conditions, but that tuberculosis is the only one present.

2. The second group of individuals to be discussed includes those cases of obvious pulmonary tuberculosis in which there are evidences of a very mild degree of silicosis or pneumoconiosis after several years of service in an industry, but in which it seems reasonable to believe that the latter condition is not a predisposing factor. By the term, 'mild degree,' is implied a very moderate nodular appearance, which may have been produced or have been found after ten to fifty years' occupation in the same industry. In many instances, it is easy to draw the line between predisposition and lack of it, but in some cases, this is a very difficult question to settle, and the value of the decision must be governed to a considerable extent by the diagnostician's ability and experience. The law in some countries gives every worker the benefit of the doubt when both conditions are present, justly so in many instances and, no doubt, unjustly in others. Unfortunately, animal experimentation is of little or no value in helping us to decide whether a very slowly progressing pneumoconiosis, requiring many years to become evident in even a minor degree, is in any way a predisposing cause of the infection, because it is impossible to obtain proof of the action of silica or other dusts when inhaled in moderate quantities

upon animals over a period of very many years Ordinarily, one recognizes two distinct types of human pulmonary tuberculosis, childhood and adult, which are, in many respects, almost different diseases. Anyone familiar with silicosis must recognize a third type, namely, silicotuberculosis, which presents many points of difference from the other two, and which will be discussed later.

In the group of cases now under discussion, we would not use the term *silicotuberculosis*. In these individuals, one would expect to find the usual appearances of adult tuberculosis, throughout its course. In many instances, we have noted evidences of a tendency to heal with only the usual amounts of fibrosis. Complete clinical and roentgenological healing of such lesions would almost always imply lack of predisposition by reason of dust. Lack of complete healing may leave one in doubt as to future predisposition and change in type, whether the individual changed his occupation or not, and subsequent serial examinations would have to be the deciding factor. In this connection, it should be borne in mind that silicosis with an active infection may be a progressive condition for many years after cessation of the dusty occupation, and predisposition may become manifest a long time after the dust exposure.

Our greatest experience with the group of cases under discussion has been in connection with coal-miners The frequently alleged protection against tuberculosis afforded by coal-dust is well known to everyone interested in pneumoconiosis. In discussing this problem, we must not lose sight of the fact that, in the coal-miner, we are dealing with two different kinds of dust, and, therefore, two entirely different conditions, one, anthracosis, due to coal-dust alone and relatively harmless; and the other, best designated by the term *anthraco-silicosis*, or silico-anthracosis, recently used by Cooke,[58] and due to a mixture of coal-dust and silica. Pure coal-dust by itself, does not produce more than a mild degree

of pneumoconiosis and probably does not, in itself, or through its own effects alone, predispose to tuberculosis. If tuberculosis occurs in the pure anthracotic, it is likely to take the usual form of adult tuberculosis. On the other hand, there seems to be conclusive evidence presented by Cummins,[59] Middleton,[60] and others, that coal-dust aggravates the action of silica when the latter is inhaled in large amounts, but Mavrogordato,[61, 62] Heffernan,[63] and Heffernan and Green,[64] and others, believe that, when the intake of silica is slight, the action of coal dust may be a retarding one on the silica. Certainly the excessive inhalation of both dusts is productive of a predisposition to tuberculosis. Cooke [58] quotes from Cummins [59] that retention of coal-dust to any serious degree does not occur unless the lymphatics of the lung have been previously damaged by the action of silica, in which case the coal-dust is capable of accumulating and leading to serious, and even fatal, disease.

3. The third group of cases for discussion comprises a very important one in which the individuals present obvious tuberculosis associated with slight evidences of silicosis, after a comparatively short period of occupation, and yet, it is most likely that the silicosis is a definite predisposing factor in the infection and its progress. There may be another factor in the silica saturated lung to be considered, which will be discussed later in connection with group 4. This present group has been a very interesting one to us and one which confused us considerably until we became oriented, because it was a new experience. We refer particularly to sand pulverizers and workers in some other industries, who are exposed to unusually large amounts of dust containing a very high percentage of silica. In the incapacitated individuals, tuberculosis was the striking feature. In most cases of this group, the evidences of silicosis were slight. The tuberculous manifestations were almost invariably those of a diffuse pneumonic process, unlike

the cases of more chronic silicotuberculosis. Moreover, the clinical aspects differed There was a rapid onset of dyspnea, cough, hemoptysis, night-sweats, asthenia, and extreme weakness, in contradistinction to the characteristic comparative freedom of the more chronic cases from serious symptoms until near the end We have no way of determining whether any of those cases had tuberculous lesions before starting work, or whether some or all contracted the disease during their comparatively short time at their occupation All of these workers were cases in which a complete knowledge and understanding of the physical factors incident to the industry were absolutely essential in the correct roentgenological interpretations.

4 The fourth group, which merits separate considerations, embraces cases of obviously advanced tuberculosis in which there are no demonstrable evidences of silicosis, or, if present, they are entirely masked in individuals working under conditions in which the physical factors in the occupation would lead one to suspect the possibility of a rapidly developing silicosis, as in group 3 The essential theoretical point is that the intake of silica has been unusually rapid, but there has not been sufficient time for demonstrable fibrosis, or even demonstrable prefibrotic stages to become established The question arises as to whether a silica saturated lung, with the preliminary stages of silicosis in progress, can act as a predisposing factor in the incidence of tuberculosis, or can reactivate old but unhealed lesions We have frequently come in contact with cases in which these questions have arisen and it is an issue that we can no longer evade.

These two aspects of the situation must be considered separately, and the second will be discussed first It would seem that the question of reactivation would have to be answered largely by the results of animal experimentation Gardner[65] states that it has been generally assumed by clinical investi-

gators that the terminal tuberculosis, which causes the deaths of at least 25 per cent of silicotic subjects, is the result of infection *acquired* during industrial life Hence, he carried out a series of animal experiments to determine whether or not certain dusts might not *reactivate* old, partly healed foci, and from this source produce endogenous reinfection Guinea pigs were infected through the air passages by the author's usual attenuated strain of tubercle bacilli, which produced the typical 'primary complex' with definite tendencies to limitation and healing, and the usual primary involvement of regional lymph nodes Groups of infected animals were dusted with quartz, granite, and carborundum dusts respectively The dusting occurred at intervals when the healing process was started, well under way, or nearly completed We are interested particularly in the effects of quartz dust, containing 99 34 per cent $SiO_2$ In the first group, when dusting began fifty-four to eighty-seven days after infection and was continued eight hours per day, except Sundays, for variable periods, there was a spread of the tuberculous process in all the animals In those exposed to dust one hundred fifty-one and one hundred seventy-seven days after infection, there was evidence of a spread in 60 per cent In the third group, in which dusting began two hundred six days after infection, 33 per cent showed spread This was usually most marked in those animals dusted for the longest period before killing The spread developed by direct extension from the primary foci with or without subsequent widespread bronchogenic dissemination

We are particularly interested in the question as to whether reactivation of tuberculous lesions can occur before it is possible for any demonstrable evidences of silicosis to become apparent Gardner's experiments would seem to prove *that it can*, but we must bear in mind that his quartz-dusted animals were exposed to an atmosphere containing 859 million par-

ticles per cubic foot under 10 micra, of which 188 million were under 1.5 micra in size, which are very high silica dust counts for human industrical exposures On the other hand, we must remember that the micro-organisms in the human subject are of much greater virulence than those used by Gardner

Let us approach the problem from the standpoint of reactivation during a prefibrotic stage of silicosis. Gardner and many other investigators have shown us, and many of us have seen for ourselves, that dust cells will phagocyte very variable numbers of particles of different dusts, and that, in the case of silica, only as few as one to four particles will be incorporated, compared to enormous numbers of coal or carborundum particles Probably, or at least, mainly for this reason, according to Gardner, quartz gives no limitations to locomotion and rapid removal to lymphoid tissue, nor does it interfere with physiological action in the sense of phagocytosis for the tubercle bacillus. It is also known that a reactivated tubercle has included in its structure great numbers of dust cells. Gardner states that it is uncertain just how the dust reactivates the quiescent tuberculous focus. It has been proved that inflammatory reactions, specific in the case of the frequent incidence of Friedlander pneumonia, and nonspecific through the applications of irritants, are not the factors *per se*.

Gardner [65] concedes that, in view of our present knowledge, or lack of it, the action must be chemical, either by silica or some other substance He regards two general views as tenable : (1) That silica may injure the tissues and produce an environment particularly suitable for the growth of the bacillus through the elaboration of some substance by the action of silica on the body cells and better suited to the micro-organism than its environment in the tubercle protected from body fields by a wall of hyaline fibrous tissue ; and (2) that in dilute solution, silica stimulates proliferation of the bacil-

lus, although we have as yet no accurate information as to such solubility in body fluids

The second aspect of the situation, in regard to the newly silica saturated lung acting as a predisposing factor in the *incidence* and rapid progression of a tuberculous infection, is not so easily disposed of. The foregoing remarks on animal experimentation would certainly apply to the rapid progression of the infectious process when once it had been initiated Predisposition under the circumstances imposed has not been proved, but it is an issue which must be met We are inclined to regard it as a strong possibility as yet unproved The type of case discussed under group 3 would lead one to suspect the possibility from both clinical and roentgenological stand points

As to the roentgenological appearances in such cases, whether the infection was reactivated or incidental through chance or predisposition, we might expect the usual rapid forms of adult tuberculosis or a rapidly progressing and spreading pneumonic process, such as were encountered in many instances in group three cases The important deciding factor in predisposition for the incidence of infection or in the silica saturation's being the cause of reactivation and progression cannot be the roentgenological examination, because the latter does not show evidences of silicosis The decision, for the present at least, must rest almost solely upon the actual and *proved* physical factors present in the individual's occupation, which must be beyond all shadow of doubt before condemning a man in this group or placing the responsibility

5 : The fifth group comprises those cases of obvious tuberculosis in which there is evidence of silicosis which could readily be a predisposing factor in the incidence of the infection This group embraces a large number of cases, and has received wide recognition The peculiar feature of this

group is that the roentgenological evidences of the infection are usually out of all proportion to the clinical aspect of the cases. These cases are well recognized as one of the group of silicotuberculosis The rapidity of progress of the infection is often, if not usually, commensurate with that of the silicosis. In the more slowly developing cases of silicosis, the infection behaves differently from ordinary tuberculosis. As Gardner [66] states, the clinical study of human pneumoconiosis complicated by tuberculosis has shown that the disease tends to run a chronic course, and the symptoms of intoxication are not so pronounced as in undusted individuals. The slow progress is due to the fibrosis, which is usually intensified by the combination of the two conditions, as is generally recognized. Gardner regards the lymphatic obstruction of the silicosis as sufficient explanation for the lack of toxic symptoms Obviously the more rapid the progression of the silicosis, the more likely is the infectious process to be influenced like the cases of groups three and four.

6 The last group does not so much concern a definite group of silicotuberculous individuals as it does a pathological problem Frequently, when examining silicotic suspects, we encounter cases whose roentgenograms show very obvious calcifications in the pulmonary lymph nodes, with or without a calcified primary nodule, and the question arises as to the possible effects of childhood tuberculosis upon the progression of silicosis In 1925, Gardner [66] stated that the lungs of individuals whose pulmonary lymph nodes were involved with healing or healed tuberculosis would more readily react to smaller amounts of dust, and that an obstructive fibrosis would occur in a shorter time He intimated that this might account for the great irregularity in the time required for the development of demonstrable silicosis in industry We believe this still remains a theoretical problem, but we feel that an *individual with a homogeneous calcification* of the

lymph nodes is a safe subject for employment mosphere, whereas the individual having calcifi are punctate or mottled may not be.

Tuberculosis is a very frequent complication and, as such, is almost certain to be the essential cau disability and is quite likely to be the eventu It is to the interest of the employer, therefore, to tain that hygienic surroundings under living, as we working, conditions are kept healthful It is highly that the worker in hazardous dusts does not contract losis from an outside source if it can be prevented. N the individual with active tuberculosis should not ployed, and, with past evidence of an infection, emp should be considered with the utmost caution

### COMPLICATIONS IN SILICOSIS [100]

*Primary Bronchogenic Carcinoma* — Primary bro genic carcinoma is arousing much interest in connection pneumoconiosis because of the possibility of the latter dition's acting as a predisposing cause (Figure 26) publicity of the historic and very obvious predisposition of Schneeberg miners to lung cancer and the more recent kno edge of a similar state of affairs among the Joachimstal und ground workers has led to a belief that pneumoconiosis m play a part in the etiology of bronchial carcinoma

We have studied a few such patients, yet we are not co vinced that there is ample justification at the present time fo a belief that pneumoconiosis is a predisposing factor in the incidence of primary bronchogenic carcinoma and for the following reasons

1 We have had the opportunity to study a large number of cases of pneumoconiosis from various industries and, in addi tion, have been so situated, in a medical center and in an institution well equipped with an active bronchoscopic clinic

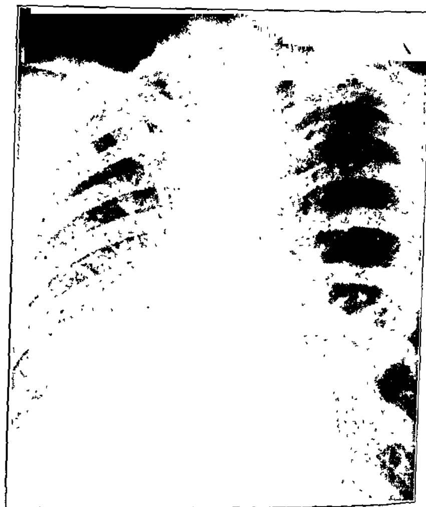


FIGURE 26

Primary carcinoma of the right lower lobe in a worker. There is very little, if any, roentgen evidence of silicosis The silicotic changes, however, were demonstrable in the post-mortem examination.

as to have referred to us for examination or consultatic many cases of primary lung cancer. Notwithstanding thes facts, we have seen only three proved cases of bronchogenic carcinoma in association with pneumoconiosis. While Weller[67] states that other authors have reported cases among coal miners, sandstone workers, cigar makers, and in many other occupations, we cannot accept isolated reports as conclusive evidence, especially as every male patient with cancer of the lung has, presumably, had a job of some kind.

2. The Schneeberg and Joachimstal cases were obviously due to some factor different from the usual causes of pneumoconiosis, and radium emanation or arsenic would seem to have been the most likely of the unproved predisposing causes.

3. While animal experimentation, as carried out by Willis,[68] Willis and Brutsaert,[69] Murphy and Sturm,[70] and others, has suggested that certain cell hyperplasias producible through the action of dusts, tar, and other irritants, may be precancerous conditions, there is no absolute proof that they are. Moreover, similar hyperplasias are noted bronchoscopically quite often in the human subject and are invariably reported histologically as inflammatory in origin. The Jacksons[71] report that, on many occasions, they have removed tumor-like bodies obstructing a bronchus and the histologic examinations have shown nothing except chronic inflammatory tissue. These growths have been regarded as serious because of the resulting secondary effects of atelectasis, drowned lungs, suppuration, abscess, and bronchiectasis. The etiologic factors have been regarded as stagnation of secretions, especially of a purulent character, and the specific granulomas — tuberculosis, syphilis, and fungus infections. Any inflammatory process in the bronchi may be associated with a nodule that assumes tumor-like form, and mechanical contraction through elongation and shortening of the bronchi during respiration

might mold protuberant inflammatory new formations into tumor-like form.

Fried,[72] like many others, is inclined to ascribe bronchogenic carcinoma to infections and inflammatory conditions. Of the three types of cells found in the bronchial mucosa-ciliated, goblet and 'basal' epithelial cells — the last is concerned in the regeneration of respiratory mucosa. Metaplasia of these cells occurs in numerous bronchopulmonary diseases, as influenzal pneumonia, measles, diphtheria, and whooping cough, and Fried regards this metaplasia as, in a sense, a precancerous state. He believes that only the basal cells produce cancer. This opinion is not shared by others. Fox,[73] for example, found 3 cancers with mucous producing or goblet cells among 37 positive biopsies in 85 referred bronchoscopic tissues.

A brief review of the Schneeberg and Joachimstal situations seems worth while for a better understanding of the relations between lung cancer and mining operations. Weller [67] states that cancer among the Schneeberg miners 'is probably the most extraordinary and at the same time the least understood of all associations which have been discovered to exist between occupation and the incidence of neoplasm.' Literature on the subject of the high mortality among the underground workers of the Schneeberg cobalt mines in Saxony began as early as 1500. Weller refers to the report of Thiele, Rostoski, Saupe, and Schmorl [74] of official investigations of 154 miners during a period of nearly four years, during which time 21 died, and 13 of these with a diagnosis of primary lung cancer made at autopsy. Two of these men had not worked in the mines for many years, which, we believe, makes the theory of radium emanation as the etiologic factor a debatable point, although it may not exclude it. Weller also refers to the investigation of the mines by Rostoski, Saupe, and Schmorl.[75] There was much hard rock

drilling and a great amount of dust. The ore contained iron, bismuth, tin, zinc, lead, manganese, uranium, cobalt, and nickel, chiefly in combination with sulphur and arsenic. Because of the uranium content, and therefore radium also, the ore was radioactive, as was, also, the mine atmosphere up to as high as 50 mache units. The possible etiologic factors considered were silica, chemically active dusts, especially arsenic, and, in volatile form, radium emanation and the flora of the damp mines. Many of the men had demonstrable silicosis. The exact factor still remains uncertain. In regard to the radium emanation, Martland[76] states that the active deposit present after death of the individual would be entirely too small to be measured.

At Joachimstal, in Bohemia, and 30 kilometers from Schneeberg, are the mines which have been famous as a source of radium, although originally they were worked for silver, cobalt, nickel, bismuth, and arsenic. The mining for uranium preceded the discovery of radium. In 1929 and 1930, Pirchan and Sikl[77] investigated these miners, who also had been known to have a high mortality rate for pulmonary diseases. During that period, 19 miners died, 10 recently active and 9 pensioners. 13 of these were examined post mortem, and primary lung cancer was found in 9. Tuberculosis, pneumonia, and trauma were the other causes of death. Unfortunately, careful studies were not made from the standpoint of the etiology of the cancer. The atmosphere of the mines had approximately the same radioactivity as at Schneeberg. Unlike Schneeberg miners, these men presented no marked degree of silicosis. We cannot help but feel that radium emanation and arsenic must be strongly suspected as etiologic factors in both of these mines, although it would be most difficult to obtain positive proof for the implication of the former.

*Cardiac Lesions.* — A few articles in the literature call at-

tention to silicotic fibrosis as an etiologic factor in cardiac enlargement and break-down. While this may be true, it is very difficult to prove, and one raises the question as to whether there may not be some other etiologic factor operative coincidentally. If such were true, silicosis might be regarded as a major factor without sufficient evidence.

## THE ERYTHROCYTE SEDIMENTATION REACTION IN SILICOSIS

There is some evidence[78] to show that the sedimentation rate in silicosis is increased. I have not had sufficient experience to allow me to state whether it is increased in individuals without active infection. Preliminary observations would seem to justify the following statement, however In the absence of clinical or roentgen evidence of active infection, in workers showing roentgen evidence of silicosis or of silicosis with healed infection, I have rarely found an increased sedimentation rate If this observation is subsequently substantiated, the sedimentation rate will become an excellent test to use in conjunction with other clinical studies to evaluate the activity or quiescence of a given lung lesion.

## ASBESTOSIS

*The Asbestos Industry.*[49] — This industry has grown extensively during the past twenty years. This has caused attention to be centered around it as a factor in industrial disease, mainly, however, because of some very unusual features found in the lungs of those exposed to the inhalation of the dust The first important one is the fact that pneumoconiosis produced in the workers apparently by silicates instead directly by silica. This fact will, no doubt, lead to furth interesting studies of industries in which silicates predo nate in the dusts. There is some confusion in regard to action of silicates in the production of pneumoconiosis

pecially as certain of them, like clay and kaolin, are supposed by some authorities to retard the action of silica In addition to these, talc, French chalk, soap stone, pumice, and many other silicate substances are extensively used in various industries Another unusual feature of pneumoconiosis in asbestos workers is, first, the ante-mortem and autopsy finding in the lungs of some very interesting, unusual, and characteristic mineral deposits, and still another is a progression of fibrosis and roentgenographic appearances differing from those of most injurious dust producing industries Numerous articles have appeared on the subject during the past four years. The most comprehensive one has been that of Merewether and Price,[79] supplemented by Merewether,[80] in 1930, and again in 1934,[81] in a general review of all phases of the asbestos industry in Great Britain and a resume of the results of their examinations of 363 asbestos workers Prior to 1928, there were available records in medical literature of only 2 deaths among employees in the factories, but since then, a considerable number have been reported The importance of the condition and its differences from other forms of pneumoconiosis seem to warrant a general acceptance of the term, 'asbestosis.'

Lynch and Smith,[82, 83] in their reports, have been the first in this country, at least in medical literature, to deal specifically with the subject One of their cases, a negro, died as a result of a gunshot injury after working twenty-eight months in a mill during a period of three years His lungs showed definite interlobular, perivascular, and peribronchial fibrosis, and also the various asbestosis bodies The second case, also a negro, died of lobar pneumonia after four and a half years of continuous work in a mill His lungs showed, everywhere, a definite increase of young fibrous tissue, in addition to the various asbestos bodies

In view of the meagre number of complete autopsy re-

ports in this country, the following report of Lynch and Smith[83] is given in its entirety:

*Autopsy*

The body had been embalmed prior to the autopsy. There was marked subcutaneous edema, especially of the feet and legs to above the knees, and some of the hands and forearms. The toes and fingers were stubby, but there was no cyanosis. The sternum was perhaps interfered with by the embalming. The sternum was flattened above and definitely depressed over the epigastrium.

The pericardium and heart were definitely displaced to the left, the apex well outside the nipple line at about the fifth interspace. The heart weighed 343 gm. empty, was definitely broadened but not lengthened, the right heart furnishing the breadth, the left, not enlarged. The right cavities were well enlarged and open, the left ventricle closed. The right myocardium measured 8 to 10 mm. in thickness well up from the apex toward the pulmonary orifice. Microscopically the muscle fibres were not of uniform size, and there was generally a distinct enlargement of those of the right. They were of indistinct outlines and striations, and those of the right ventricle were frequently vacuolated. There was an increase of stroma, and the veins, particularly of the right, were definitely distended with blood.

The aorta was the seat of a minor atheroma, and the whole vascular system, outside the heart, was in good condition.

Both pleural sacs were completely obliterated by old adhesions, usually very dense, especially over the whole right lung and the upper and lower left. The diaphragm was up to about the fourth rib on the left but was low on the right. The left lung was retracted well over to the outer wall and high, occupying about one-half to two-thirds of the usual space. The pleura generally was thick and cartilage-like, especially over the apex and base. The lung was coarse,

leathery, nodular and lumpy over the upper half and the base, the middle being more spongy and air-bearing. The vessels and bronchi were very prominent emphysematous bullae in the parenchyma especially in the apex where there was a group or chain of honey-combed sacs. The interlob pleura was obliterated The bronchi appeared congested. Th hilum lymph nodes were inconspicuous, did not appear en larged, and were smoky black There was no evidence o tuberculosis

The right lung was large, filling the whole right chest and encroaching some toward the left Its whole pleura was very thick and cartilage-like The interlobar pleura was sealed outwardly, but was open, a clean membrane presenting, between the adjacent lobes The lung was very much like its fellow generally, the upper lobe densely fibrous and lumpy, vessels and bronchi prominent, and a mass of emphysematous bullae in the apex The middle lobe was not so fibrous, nodular or lumpy, and had prominent bronchi and air bullae. Both lungs, especially the right, bore much frothy fluid, which, expressed and examined, revealed numerous asbestosis bodies, free, in sheaves or clumps, and ingested by giant cells The two ends were often engulfed by different giant cells There were also a few dust cells, with fine black granular pigment, and masses of a yellowish amorphous substance, of the color of the asbestosis bodies, in cellular debris

Microscopically there was an extreme grade of hyalinizing fibrosis of the lungs, universally but irregularly distributed The pleura was thick and fibrous, and there was marked interlobular fibrosis Scattered here and there were irregularly rounded areas of hyaline fibrous tissue, in somewhat laminated form within which were masses of greenish black granular substance Here were also areas of liquefaction and calcification in the centre of these hyaline nodules A large part of the alveoli were obliterated or virtually so Some

lobules remained open, the sacs having thick fibrous walls In these open alveoli, the epithelium was sometimes cuboidal and there were fairly numerous large round phagocytes, some with a group of nuclei, some mononuclear. These macrophages contained black or greenish-black or brownish granular pigment and an occasional asbestosis body. Where the lung was less fibrous, there were young connective-tissue cells and lymphocytic accumulations. The bronchioles were dilated and their walls thick and fibrous. In some areas, the lobules of alveoli showed marked emphysema, large empty sacs with thin walls Asbestosis bodies in typical forms, with a variety of architectural figures, yellowish-brown, clubbed, dumb-bell and rod forms, were to be found widespread, singly or in groups within giant cells in the alveoli of less fibrous areas, singly in the alveolar walls and interlobular tissues showing the younger fibrosis Associated with them was much granular substance of the same color, as if from disintegrated asbestosis bodies. Besides these pigments, there was much ordinary black anthracotic material around the vessels of the interlobular tissues The interlobar pleura was especially thick. The large bronchi were practically normal The peribronchial lymph nodes were the seat of marked fibrosis, edema, atrophy of follicles, and accumulation of masses of black and yellowish-brown granular pigment, of the same order as that in the lung.

The liver was grossly and microscopically in a state of extreme chronic passive congestion, which state, in lesser degree, was conspicuous in spleen, kidneys, and other viscera

More and more data of this type are becoming available as recorded in the more recent contributions of Merewether,[83] Lanza, et al.,[84] Lanza,[85] and Shull [86]

*Symptomatology* — The symptom complex of pulmonary asbestosis may be of interest to roentgenologists, especially because the condition is a rather new one in the group of

pneumoconioses, or, rather, an old one come to light and extensively studied. We have made a composite grouping of symptoms assembled from the reports of Wood,[87] of his 15 cases, Haddow,[88] Stewart and Haddow,[89] Soper,[90] and Wood and Gloyne,[91] including 22 cases additional to those reported by Wood, as follows:

(1) *Onset.* This is variable and depends upon the exact occupation and the quantity of dust inhaled. Both are important, for the individual may not be engaged in a very dusty duty, but may be working in a room made very dusty by another procedure in the manufacturing process. Wood states that symptoms may appear comparatively early and gives, as the time limits, one and fourteen years. Haddow places the average period at about five years, and states that the condition is usually recognized first during or after an attack of influenza or winter cold. Cases are usually better in summer.

(2) *Cough.* This is a very variable symptom, sometimes nearly or entirely absent and seldom excessive.

(3) *Expectoration.* This is moderate when present, but is usually lacking except during bronchitic attacks.

(4) *Anorexia.* This is a rather constant late symptom. Haddow regards it as an indication to stop work. Individuals may then live for several years, but become progressively weaker, more emaciated, and exhausted, until pneumonia or bronchitis brings death.

(5) *Dyspnea.* This is the most striking symptom and practically the most important one. It is progressive as in other forms of pneumoconiosis, whether the individual stops work or not. Wood and Gloyne speak of it as "a terrible tightness of the chest," which is very expressive. While dyspnea is due primarily to inelasticity of the lungs, which is always beautifully demonstrated by roentgenoscopic observations of the diaphragmatic excursion, it is also, in a measure,

a result of interference with blood supply. Therefore, it is slow and insidious in its development, until, finally, the lungs are able to accomplish no more than just sufficient oxygenation of the blood to sustain life.

(6) *Cyanosis* This is a very frequent late manifestation

(7) *Weight Loss and Emaciation.* These manifestations are very striking in the late stages. They are out of all proportion to the physical signs, and thus differ from tuberculosis alone

All of these symptoms are more or less those found in other forms of pneumoconiosis, and a symptomatology largely applicable to all forms is placed here for emphasis on this particular one. The last three symptoms are to be emphasized in connection with the same pulverizing industry.

There is an interesting skin lesion found in connection with the asbestos industry known as the asbestos corn It has been mentioned particularly by Gloyne and by Soper, among the writers on pulmonary asbestosis The fine asbestos fibers penetrate the superficial layers of the epiderm like *thistle thorns and produce small corns or hypertrophies of* the epiderm around them. No asbestosis bodies have ever been found in these lesions.

*Roentgenologic Considerations.*[49] — Comparatively few of the report on pulmonary asbestosis contain roentgenographic findings, and in many of those which do, the descriptions are not so scientifically made as to permit of easy comprehension or interpretation Most writers who describe the appearances or mention them casually seem to regard them as most unusual or characteristic of the occupation, notably Wood,[87] Wood and Gloyne,[91] and Merewether [80] It is true that they are unusual, but we would hardly regard them as characteristic for the reason that other conditions induce quite similar roentgenographic appearances In all industrial pneumoconioses in which the roentgenographic appearances deviate

from the stereotyped description first given to those of the hard rock miners, one must become familiar with those of the industry and correlate them with diaphragmatic excursion, clinical findings and degree of disability represented before being able to interpret the findings for that particular industry For example, from the roentgenograms of 17 asbestos factory employees, examined by Pancoast in 1917 and reported by us in 1926,[92] the appearances were not regarded as of any particular moment, but simply as minor changes such as are frequently found in connection with slowly fibrosing dusts On reviewing these roentgenograms with Dr J. V Sparks,[93] who made the roentgenological studies of Wood's cases, referring to our records of the diaphragmatic measurements, and with a knowledge of the peculiarities of the asbestos industry, particularly from the standpoint of roentgenographic appearances, we at once realized that our first interpretations were based upon an entirely erroneous conception It is questionable whether anyone viewing a number of roentgenograms of atypical pneumoconiosis representing several industries, could pick out the ones of asbestosis subjects alone All of the cases represented in the series just mentioned were only moderately advanced In some roentgenograms of asbestos factory workers seen with Dr Lanza,[94] we noted that there was a direct progression into a terminal diffuse fibrosis without any nodular predominance This terminal state seemed to have been reached in about twenty years, on an average Nevertheless, in some instances, the nodular fibrosis was quite evident

Wood[87] has given a very excellent description of the roentgenographic findings in the 15 cases examined, and this has been corroborated by Soper[90] in 1 case, by Oliver[95] and by Wood and Gloyne[91] Our interpretation of these findings was that there had been a rather predominant early interstitial fibrosis, with, later on, an associated soft nodular fibrosis

The transition to the third stage, if we should adhere to that term, or at least to the terminal stage, would seem to have been mainly through a continuance of the interstitial predominance with very little prominence of nodular fibrosis. The early interstitial predominance was described as resembling a ground glass appearance.

On reviewing our own roentgenograms with Dr Sparks and comparing them with the excellent roentgenograms of some of Wood's cases, we found a great similarity in the appearances. The early evidence of the condition was a faint homogeneous haze in the lower part of the mid-lung field which seemed to correspond to the appearance we have previously suggested as an early predominance of interstitial fibrosis This haze spreads through the lower one-half or two-thirds of the lung field and may almost obscure the domes of the diaphragm later on, with very little evidence of nodular fibrosis. With it, there is a very decided restriction in movement of the domes of the diaphragm without the appearance of pleural adhesions, which seems to be proof of the general fibrotic nature of the process. Thickened pleura does exist, of course, as in any fairly well advanced case of pneumoconiosis. The most unusual feature of the condition seemed to us to be the fact that, in both sets of roentgenograms, the appearance seemed to begin more often on the *left* side than the right, and progressed more on that side This is the reverse of all other occupations that we have studied Merewether [80] states that it begins on the right side, as one would expect, and the cases we observed may have been examples of the exceptions which are occasionally observed in any industrial pneumoconiosis We noted the same tendency to obliteration of the left cardiac border as mentioned by Wood, because of the diffuse fibrosis and the prominent trunk shadows Wood states that the late stage may or may not show consolidations Wood and Gloyne [91] state that

the ground glass appearance in late cases, on careful analysis, resolves itself into a fine mottling and linear shadows which, toward the base, may look like cobwebs.

It is evident that the nature of these changes in the lungs requires the best type of roentgenograms for their portrayal. Also, the seriousness of what may seem to be minor changes to the uninitiated requires a roentgenoscopic study of diaphragmatic movements to make one realize the actual state of disability present. It is evident also, as Merewether states,[80] that an opinion as to the degree and intensity of an asbestosis fibrosis, based upon a comparison of roentgenographic changes with those shown in standard silicosis films, will be an underestimate.

More recently, Merewether[81] and Shull[86] have not emphasized on which side the process is most evident in the beginning or late in the disease. This coincides with my more recent experience. The disease process may be diffuse, bilateral, or unilateral, but is practically always limited to the lower half or lower two thirds of the lung field.

Of the 363 cases examined by Merewether and Price, 133 had roentgenographic studies. Of these, 52 were said to have presented appearances of diffuse fibrosis and 22 were suspicious.

Merewether[81] says that approximately seven years must elapse between the commencement of exposure and the production of a serious degree of asbestosis. This space of time includes not only the trapping period of the fibrosis producing dose of dust, but also the maturation period of the fibrosis, which periods, of course, overlap. The existence of this fibrosis-producing period accounts for the fact that no appreciable number of asbestosis cases are discovered until the second five years of employment. Relatively few cases mature in this minimum period of seven years; many, even in the more dusty processes, require eleven years. When

fibrosis of serious import has matured, the worker is unduly short of breath on any extra exertion, has a slight cyanosis of the lips, and a little dry cough, mostly in the mornings. He still, however, is quite able to work and, usually, is not concerned about his state of health. Many workers, in fact, will not admit symptoms of any kind at this stage.

*Discussion of the Nature of the Roentgenologic Appearances seen in Asbestosis.*—From my experience, the roentgenologic findings in asbestosis are largely limited to the lower half or lower two-thirds of the lung fields, whereas in silicosis, the opposite obtains. In asbestosis, the lesion may be bilateral or largely unilateral. Emphysema, blebs, or bullae always accompany well established changes in asbestosis and occur in the upper portions of the lung fields; whereas, in silicosis, the lower lung fields, to a large extent, are the site of these changes.

I have found it extremely difficult to determine the early manifestations of asbestosis. I believe an explanation for this would include the lack of an adequate pre-employment examination, in which a careful fluoroscopic study of the respiratory and cardiac movements is made. It seems to me that fluoroscopy, directing special attention to costal and diaphragmatic expansion, is essential in the determination of early changes in asbestosis.

If one can assume that the pathological process is due to changes secondary to the lodgment of asbestos fibers in the smaller bronchioles, the roentgen findings might be explained as follows. Disturbance in the roentgen appearance would not be seen except following the lodgment of the fiber in the smaller bronchioles. Sufficient time would have to elapse until a definite collar of fibrosis had become manifest so extensively that it would limit the ingress and egress of air into the lung areas distal to the point of obstruction.

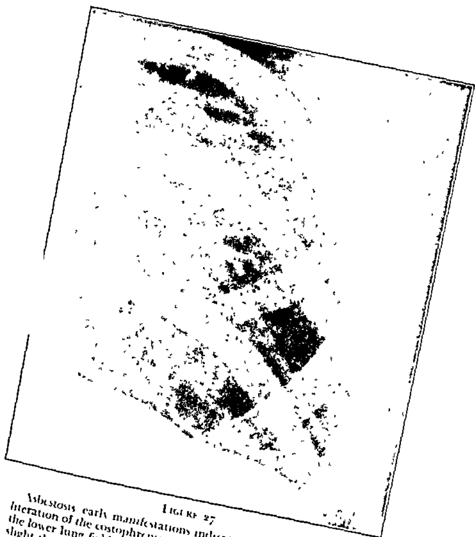


FIGURE 27

Asbestosis early manifestations indicated by the beginning obliteration of the costophrenic sulcus. Moderate hypoventilation of the lower lung fields due to thickening of the parietal pleura and slight thickening of the interlobar pleura.

In such damaged bronchioles, it is justifiable to anticipate that the lack of ventilation and drainage, distal to the point of obstruction, might become an ideal site for the development of infection or atelectasis. The infection, in turn, would increase the amount of serum protein in the tissues and assist, or stimulate, a pre-existing tendency for fibrosis. The fibrosis, itself, might limit the amount of normal lymph flow and, in succession, the infection would extend to and involve neighboring areas. When a sufficient amount of tissue had become involved, one might be able to demonstrate beginning changes in the roentgenogram, such as beginning obliteration of the costophrenic sulcus and fluoroscopically, some limitation of costal and diaphragmatic expansion. (Figure 27) I should assume, when such an appearance is observed in the roentgenogram, that the process might be regarded *as moderately advanced.*

The next step in the pathological process would seem to include an extension of the affection to the periphery. *With involvement of the periphery, there is a definite limitation* in the respiratory movements, which would be extremely slight in the early case of asbestosis but marked in the more advanced degrees of the disease. (Figures 28 and 29) The ribs are somewhat fixed and in thin individuals, they are dependent. In fat persons, they change very little. The interspaces may become quite narrow.

In the consideration of the healthy chest above, I called attention to the importance of the respiratory movements in lymph flow, and also to the fact that any restriction of these movements would tend to limit the flow of lymph Any limitation of the flow of lymph increases the development of pre-existing pathologic changes It was, likewise, emphasized above, that thickening of the pleura occurs to a greater extent in the lower thoracic region than it does in the upper, the explanation for this being the powerful costal and dia-

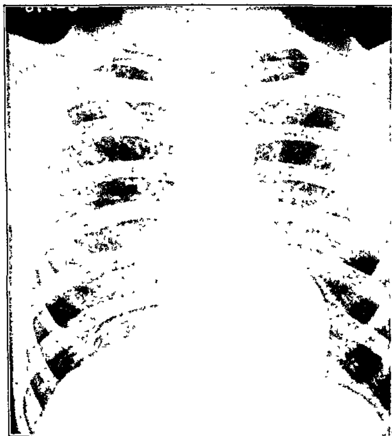


FIGURE 28

Man aged 31, silk weaver for twenty years, asbestos weaver for five years. The roentgenographic findings are within the limits of a healthy chest.

phragmatic movements in the lower chest and the relative fixation of the upper chest.

Almost every autopsy protocol has called attention to the

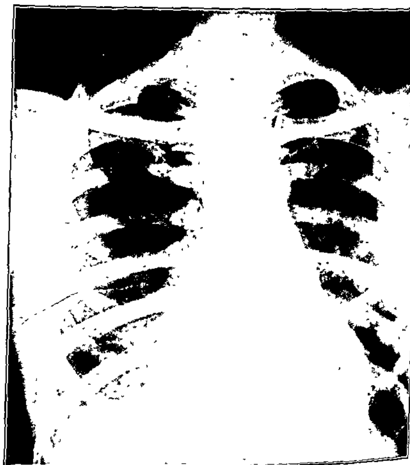


FIGURE 29

Asbestosis moderately advanced Same individual as illustrated in Fig 28. Examination made five years after that illustrated in Fig 28 Note the relatively marked changes in the right lower lobe, which include thickening of the parietal and diaphragmatic pleura, obliteration of the costophrenic sulcus, slight thickening of the interlobar pleura, some narrowing of the interspaces and enlargement of the cardiac silhouette. There is evidence of hyperventilation in the upper lung fields.

marked thickening of the pleura and occasional thickening of the pleuropericardium. It is my feeling that these definite changes in the pleura can be adequately explained upon the basis of an underlying infection of the lung, extending to the pleura with subsequent thickening of the structure, due to the powerful respiratory movements and cardiac pulsations (Figures 30 and 31)

The haziness of the lung fields, called attention to by almost every writer, can be explained by pleural thickening and a lack of ventilation. One should be careful in distinguishing between the shadow caused by pleural thickening and that resulting from the subcostal muscles This has been discussed under the healthy chest

I have been forced to place less and less importance upon observations concerning the trunk or vascular markings These shadows are so changeable with variations in phases of respiration and cardiac pulsations that, unless one is re-examining patients and employing critical technics, such as making exposures during forced inspiration and during the systolic or diastolic phase of the cardiac cycle, one is absolutely confounded with changes in the size of the shadows entirely within physiologic limits

However, when changes develop sufficiently, one can see disturbance in the movements of the domes of the diaphragm and of the ribs, and obliteration of the costophrenic sulci From this point on, the pathologic process in the lungs seems to advance fairly rapidly, exactly as one would expect it to do This can readily be demonstrated by comparing the vertical diameter of the thorax in serial examinations of an individual who has a well developed asbestosis If a period of at least five years is allowed to elapse, the vertical dimension is materially reduced This observation corresponds to the limitation of the movement of the domes of the diaphragm, and, in a rough way, indicates the ability of the individual to

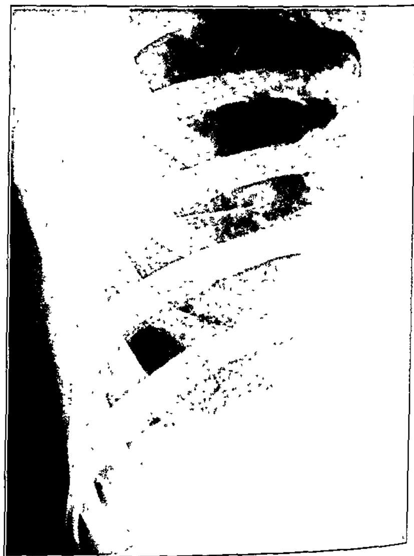


FIGURE 30

Asbestosis moderately advanced Close-up of the right lower lung field in the individual illustrated in Fig 29



FIGURE 31

Asbestosis moderately advanced. Note the position of the ribs,

their identity



FIGURE 30

Asbestosis moderately advanced Close-up of the right lower lung field in the individual illustrated in Fig 29

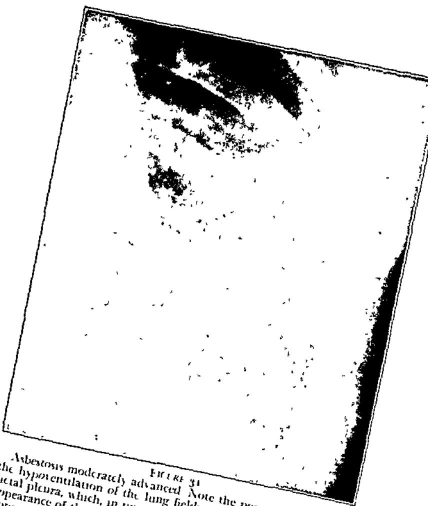


FIGURE 31

Asbestosis moderately advanced. Note the position of the ribs, the hypoventilation of the lung fields and thickening of the pa-
rietal pleura, which, in turn, are responsible for the ground-glass
appearance of the lung fields. The interlobar pleura between the
upper and lower lobes is thickened. From its position, one can
readily see the extent of scar contraction already existent in the
right lower lobe. The vascular trunk shadows have largely lost
their identity.

ventilate the lung fields. The shadows are more pronounced in the bases and diminish as they approach the upper third of the lung field.

FIGURE 32

[illegible] advanced. The lesion occupies the lower [illegible]

[illegible]

posed shadows in the lung fields. Note the [illegible] the ribs.

My comments thus far have been limited entirely to the lungs and pleura. The heart and pericardium also suffer in asbestosis, much more so in this occupational disease, in fact, than in any dust hazard with which I am acquainted. With a moderately advanced degree of asbestosis, the shadow of the cardiac outline is obscured by the shadows of superimposed lung structures and involvement of the pleuropericardium (Figure 32) One rarely sees the cardiac shadow sharply demarcated, as observed in ordinary roentgenograms The pleuropericardium becomes thickened and, in some instances, markedly so, for the same reason that the pleura over the lower lung fields and the domes of the diaphragm become thickened Pulsations of the heart and infections extending to the pleura in this region play a major role, I believe, in producing the marked thickening of the pleuropericardium These changes and those of the lung probably account for the right-sided hypertrophy and ultimate decompensation of the heart. In some instances, the cardiac silhouette is quite enlarged Realizing, therefore, that ventilation of the lungs is seriously interfered with and that cardiac action is impaired, one can readily understand why these patients are embarrassed with dyspnea, weakness, and cyanosis

In my present state of knowledge, if I were called upon to make a classification of asbestosis from a roentgenographic standpoint, I should include only two stages; asbestosis moderately advanced, and asbestosis markedly advanced, or asbestosis, first and second degree I feel quite certain that the roentgenologic diagnosis of the clinically early stage of asbestosis is not entirely reliable (Figure 33)

*Prognosis* — It seems to be the rather general opinion that the prognosis in pulmonary asbestosis among factory workers is to be regarded as grave without adequate protection, in a large number of cases, among those who work



FIGURE 33

Asbestosis, an unusual type This may be due to the fact that the individual has had some silica exposure The shadows in the middle third of the right lung field are not typically nodular.

where there is great concentration of dust. The condition is apparently progressive, even after cessation of occupation Merewether and Price [79] state that, with continued exposure to high concentration dust, fibrosis may be fully developed

in from seven to nine years, and that death may result in about thirteen years. With less concentration, fibrosis may not be fully developed for fifteen, twenty, or twenty-five years. Inhalation of dust in high concentration results in a more marked degree of fibrosis in a shorter time than when the concentration is lower.

There is no disease among the industrial hazards in which it is more important to have serial examinations than in the asbestos industry. Almost every investigator has been hampered by either no pre-employment roentgenologic examinations of the chest or relatively few opportunities for reexaminations of the individual exposed to asbestos dust.

Shull[86] states that it would seem that improvement could be expected in the early cases, but that, as the disease progresses, improvement is less likely. Sparks[96] feels that it is too early in the study of this disease to say very much on the subject. Lanza[85] believes that it is by no means certain that asbestosis progresses as does silicosis after withdrawal from dust exposure, nor that infection seems to be so closely and intimately associated with asbestosis as with silicosis.

My experience in this condition is also limited, but I feel quite certain that, when asbestosis can be diagnosed by roentgenologic examination, the condition is likely to become progressive, due to the important role exerted by fixation of the lung structures secondary to the pleural involvement. This can be readily demonstrated in serial roentgenograms, even in the absence of fine lung detail. The striking feature is the reduction of the perpendicular diameter of the chest.

*Predisposition to Tuberculosis.*—The condition of pulmonary asbestosis being a new child in the pneumoconiosis family, a great amount of interest centers around the incidence of tuberculosis. Among the early case reports referred to in the beginning of this occupational section, pulmonary tuberculosis was reported as an accompanying condition in a

few instances. Cooke's case had advanced tuberculosis Simson reported 1 case which died with tuberculosis and 1 without evidences of the infection He thought that we could not as yet formulate any definite opinion as to the frequency of the predisposition. Simson quoted Hoffman[97] as reporting 13 deaths among asbestos workers, 3 of whom had tuberculosis He also quoted Collis[98] as reporting 5 deaths from tuberculosis among less than 40 workers at a factory Wood and Gloyne[91] referred to 3 cases showing evidences of tuberculosis at autopsy. Haddow[88] observed many asbestosis cases, among whom 4 died. He did not believe that the condition predisposed to the infection, at least not as a rule Merewether and Price[79] stated that asbestosis differed from silicosis in the distribution of the fibrous tissue, in more rapid development, in roentgenographic features, and, possibly, in a *lessened susceptibility to tuberculosis* Merewether[80, 81] states that, out of 374 cases examined, evidence of active tuberculosis was found in 3. He thinks that, while there is no outstanding susceptibility proved, the question has not been fully settled as yet. I believe, however, that when tuberculosis does complicate asbestosis, that one can readily suspect or diagnose the tuberculous lesion with a fair degree of accuracy, due to the fact that the tuberculous process often occurs in the upper lobes, where there is usually very little evidence of asbestosis If a basal tuberculous process were superimposed upon asbestosis, it would be very difficult, if not impossible, to diagnose it. (Figure 34)

*Differential Diagnosis —Passive Congestion of the Lungs as a Result of Cardiac Decompensation* — In this condition, the enlargement of the heart shadow, the abnormalities of the cardiac silhouette, especially in cases of mitral stenosis, and the clinical picture serve to establish the presence or absence of passive congestion If asbestosis is absent, the appearances are likely to disappear with restoration of com-

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FIGURE 34

Asbestosis with a complicating tuberculous lesion in the right upper lobe It is entirely possible that tuberculosis may account for the entire appearance found in this individual, but it was subsequently shown that this individual not only had tuberculosis but also had asbestosis in the lower lung fields

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pensation, but not altogether in long-standing cases. Marked emphysema is usually associated with asbestosis and is apt to cause a certain amount of rotation of the heart, so that the sagittal roentgenogram does not convey a direct impression of cardiac size or shape. The lateral view is always necessary to clear up the diagnosis. Any phase of asbestosis is likely to have its roentgenographic appearance greatly accentuated by passive congestion.

*Advanced Bilateral Bronchiectasis.* — The dilated bronchi, retained secretions and associated tracheobronchitis characteristic of this condition will produce the appearance of greatly accentuated trunk shadows, which may simulate that of the moderately advanced phase of asbestosis.

Other conditions that simulate asbestosis include polycythemia or erythremia and infiltrating malignant metastases, especially those extending from the mediastinum.

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The author has drawn rather freely from a number of the writings in which he has collaborated, in view of the fact that their observations largely represent his present views When present opinions have differed, a discussion of the reasons for the change is included in the text

# IV. PATHOLOGY

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## INTRODUCTION

### Definitions

The term *silicosis* will be used in this chapter to denote the changes which take place in the body tissues as a result of the inhalation of dust containing silicon dioxide, generally referred to in this connection as "free silica." The International Conference held at Johannesburg[1] in 1930 proposed in interpreting this term that the following conditions be satisfied, viz, that the silica must reach the lungs (a) in a chemically uncombined condition, although it might be mixed with other dusts; (b) in fine particles of the order of less than 10 microns in diameter, (c) in sufficient amount, and over a certain period of time.

For some years pathologists regarded free silica as the only siliceous dust likely to produce the disease, but recently "combined silica" or various mineral silicates found in industrial dusts have been included in the indictment and a new term, *silicatosis*, has come into use to denote the types of disease thus produced

The chief example of this new group of diseases has already become sufficiently important to receive a special designation, *asbestosis*, a disease resulting from the inhalation of the dust of asbestos, which contains the silicates of iron and magnesium

As the diseases caused by these various substances differ

in their pathology, it will be necessary to describe them separately, but before doing so certain general considerations from a pathologist's point of view require discussion Just as a chronic infective disease such as tuberculosis is regarded from the three-fold point of view of the organism, the portals of entry and the tissue reaction, so in the study of a pulmonary dust disease the nature of the dust, the conditions of its entry into the body, and the tissue reaction which ensues all need separate consideration

*Nature of the Dust* Mavrogordato[2] (1930) has classified the dusts which enter the lung into four types (1) Dust that is harmless, (2) dust that is usually harmless but which may contribute to the lung mischief when inhaled in association with a harmful dust, (3) dust that is usually harmless and may actually have a prophylactic value when inhaled in association with a harmful dust, (4) dust that is harmful. The first group does not concern us here In the second group may be placed such dusts as iron ore and coal dust in association with silica The third group is a doubtful one At the moment one would not feel justified in placing any dust in this category with certainty, for the pneumoconioses take many years to develop and surprises are not uncommon The fourth and last group must include nowadays not only free but also combined silica

*Free silica* occurs mostly in the form of mineral quartz In the British Silicosis Compensation Scheme of 1928 it is referred to as silica rock, quartzose sand or 'any dry deposit or dry residue of silica'

*Combined silica* includes a large number of silicates which tend to increase with industry The commonest of those in use which are under suspicion as possible producers of pulmonary changes are enumerated by Middleton (1936)[20]

*The size of the dust particle* is important. Only very small particles reach the lung tissues Probably the majority, as

Mavrogordato suggests, are about the size of the common pathogenic micro-organisms.

*The shape of the particle* has also a bearing on the resultant lesion Broadly speaking there are 3 morphological types. (1) *Small particles with irregular surfaces and sharp* edges and diameters approximately equal. This is the common shape for quartz particles. (2) Long fibres of asbestos, which may extend up to 100 microns in length. Even if we regard the particle as inert, size and shape are of great importance because they determine the penetrating power of the particle The long needle-like asbestos fibre readily "engages" in *the respiratory bronchiole and easily comes to rest* athwart the lumen of any narrow passage it may enter, whereas the small silica particle whose diameters do not differ greatly can readily penetrate to the peribronchial lymph spaces and the mediastinal glands. (3) Minute needles of fibrous sericite — a hydrous silicate of aluminium and potassium. These vary from 2 to 5 microns in length and have been described by W.R Jones[3] (1933) in silicotic lungs as occurring in 'piled-up bundles of needles,' hundreds of which are present, he states, for every grain of quartz that can be recognised Although sericite had been previously described, it was not regarded as of great importance until the work of Jones He regarded it, rather than the uncombined silica or quartz, as the important causative factor in silicosis At the present moment the matter must still be regarded as sub judice. Obviously a series of carefully controlled pathological observations is needed in workers who have been exposed to the dust of quartz particles alone and *sericite particles alone, but according to* Drinker and Hatch[4] (1936) quartz free fibrous sericite is difficult to obtain. Middleton has pointed out, however, that in Britain workers exposed to sericite without uncombined silica are found in

the china clay industry and that so far there has been no high incidence of silicosis in this industry

The experimental evidence in laboratory animals falls outside the scope of this chapter

Non-siliceous particles are found in practically all cases of silicosis and may cause confusion Carbon, for example, is generally present in the shape of very fine amorphous particles, but coal dust may occur as elongated sharp jagged particles often up to 10 microns in length

In the course of recent investigations on dust sampling, Briscoe, Matthews, Holt and Sanderson [5] (1937) noted that dust which had been exposed for some time to the moist atmosphere of mines lost an unusually large amount of water on ignition, in striking contrast with the relatively small loss of weight on drying at 100 C, indicating that the dust had taken into firm combination a relatively large amount of water In other words the comminution of the minerals had exposed fractured surfaces on which, at numerous points, there existed free and unsatisfied valency forces whereby water was attracted and held as water of constitution in the hydrated particle Hydration of this nature can apparently liberate bases held in the crystal lattice The writers found for instance that alkali was liberated in amounts equivalent to about one-sixth of the total alkalis present in the minerals Obviously important new possibilities are opened up by these researches Freshly formed dangerous dusts yield to water both alkali and soluble silica in larger amounts than do 'dead dusts' — i e, dusts which have been exposed to air for some time

*The Portal of Entry* Probably only a small fraction of the inhaled dust reaches the pulmonary alveoli The greater portion is deposited in various parts of the respiratory tract and expelled therefrom In the case of asbestos workers, for

instance, the fibres can be found in the nose and throat and even occasionally in the conjunctival sac. The histological examination of the walls of the larynx, trachea and large bronchi in cases coming to autopsy usually reveals no lesions of asbestosis. Probably the same is true of silicosis though the writer knows of no investigations on the point. It may be assumed, therefore, that mechanical forces come into play to drive out the invading dust. Such are the muscular movements of the eyelids, the cleansing effect of the lachrymal secretion, the action of cilia and the outward flow of mucus in trachea and bronchi In the case of cilia these protective phenomena have been demonstrated by Leonard Hill[6] (1928). Using the trachea of horse, ox and sheep, he found that a suspension of lamp black in Ringer's solution was carried along the mucous membrane by the cilia at a rate which might be as fast as 3 centimeters in one minute and that fine particles such as those of iron and aluminium filings were readily passed along the trachea in this way. If a small area of ciliated epithelium was damaged by injury, cautery, or various chemicals (including chemical fumes) the transport of suspended particles was greatly reduced or even inhibited altogether Irvine[7] (1930) has suggested that repeated inhalation of silica dust may lead in the course of time to a dry bronchitis and bronchiolitis with denudation of epithelium In this case the portal of entry is opened wide, as it were, and the penetrating dust will meet with little or no resistance No doubt, too, some types of particles may be more readily expelled than others. These various factors may explain why different dusts seem to have a predilection for different parts of the lung

*The Tissue Reaction.* Obviously the tissue reaction must, to some extent, be dependent on a variety of factors, viz, the duration or intermittent nature of the dust exposure, the size of the dose, the admixture of dusts, the age and sex

of the worker, and the condition of the respiratory passages prior to exposure These factors take long years to observe in conjunction with employment histories and post mortem findings and in many industries the necessary data for assessment are not yet available Meanwhile the interaction of the dust particle and the tissues may be briefly and tentatively summarised as follows —

(1) Many dust particles are doubtless expelled en route, but if a particle reaches the end of the journey in the distant parts of the portal of entry it "stays put" It does not multiply or become bacteriolysed like a micro-organism but remains as a more or less insoluble and irremovable irritant particle

(2) The end of the journey for the silica particle is the peribronchial lymphoid tissue, but in the case of the asbestos fibre it is the respiratory bronchiole and the alveolus, hence the difference in the pathological picture

(3) Although bacteria may be found mixed with the dust, they are for the most part non-pathogenic organisms, the dust particle can therefore be regarded as a non-infective *irritant, and the tissue reaction is fibroblastic, ending in the* production of the collagenous fibre

(4) The amount of plasma exudation is minimal The exudative reaction in the lung is characteristic of the bacterial diseases

(5) Although we speak of the silica particle and the asbestosis fibre as being practically insoluble, many investigators believe that a colloidal silicic acid is actually formed by a slow disintegration of the particles in the lung and that the morbid processes are the result of a toxic action There is indeed experimental evidence in the work of Gye and Purdy[8] (1922) in favour of such a view in the case of silica sol Asbestos fibres are known to persist in the lungs of persons who have not been exposed to the dust for more than twenty years and any disintegrating action must therefore

be extremely slow. Sundius, Bygden and Bruce,[9] (1936) indeed, consider that this chemical theory of slow disintegration of dust particles is still an open question

(6) It follows that if the dust particle is practically insoluble or only very slowly soluble, the lung must in the course of time become choked with irremovable foreign matter This dust accumulates chiefly in the lymphatic drainage system.

(7) The process of phagocytosis, so prominent a part of the tissue reaction to bacterial diseases of the lung is much modified Many of the particles, notably the asbestos fibre, are too large to be engulfed by a single phagocyte and a group of these cells can often be seen surrounding but not engulfing the meal which is obviously too large for them. Again, the particle even if engulfed cannot be digested and disposed of in the same way as the more soluble protoplasmic bacterium This may be one reason why the phagocyte appears to remain so long in situ in the silicotic tissue in experimental animals. Lastly the phagocyte is for the most part a large mononuclear macrophage and not the small polymorphonuclear leucocyte from the blood stream. The latter is the phagocyte, par excellence, of pyogenic cocci *in the* lung

(8) Lymphoid hyperplasia. This is one of the striking features of the tissue reaction Small collections of lymphoid tissue are normally situated on the outside of the walls of the small bronchi but they are so small as generally to escape notice These minute lymphoid aggregations absorb much carbon pigment and are a striking feature in the lungs of all persons exposed to the dust of large towns irrespective of silicosis. They should be distinguished from true lymphatic glands They become markedly hypertrophied as the result of the drift of the silicotic particles into them. In the course

of time collagenous fibres are laid down and constitute the first simple silicotic nodules

## SILICOSIS

### Macroscopical Appearances

As a rule the body of the silicotic is not markedly wasted unless tuberculosis has supervened Cyanosis may be present Deformity of the chest wall is rare, whereas in the fibroid disease of the lung of non industrial origin which begins generally in childhood, flattening and deformity are common

*Lungs and Pleurae* On opening the thorax the lungs as a rule do not collapse They are heavier than normal and frequently bulky Pigment is variable in amount In the British cases it is generally pronounced owing to the carbonaceous dust of the industrial towns and may indeed be extreme in amount Emphysema is almost the rule, bullae at the apices being especially common, whilst all the free borders may be affected

The essential feature of the silicotic lung, however, is the nodular fibrosis This begins with small raised hard nodules or islets of fibrosis immediately beneath the pleura and in the peribronchial regions of the lung The former can be felt by running the fingers lightly over the pleura and are less pigmented than those in the lung At first the nodules are no bigger than miliary tubercles but as the disease progresses adjacent nodules coalesce into composite nodules or small irregularly shaped masses which look rather like pieces of hard black rubber embedded in the lung They can be detected by the touch as the knife cuts through the lung substance and they stand out prominently on the cut surface as the crepitant lung tissue collapses, but a true 'gritty' feel is

only imparted to the knife, as a rule, by extreme cases such as the stone mason's lung. In this case small particles of quartz can actually be dug *out of the nodule with the point* of the knife The nodules are usually more plentiful in the upper lobes where they readily tend to run together into masses of large size.

As the presence of the nodule is the essential feature for diagnosis, attempts have been made to arrive at a classification in terms of distribution and severity of the lesion. Simson and Strachan [10] (1930) in describing simple silicosis as it exists in the South African mines give the following classification which is used by the Miners Phthisis Bureau ·

(1) Slight — lesions small and moderately numerous, or medium-sized to large but sparse;

(2) Moderate — lesions numerous and small or moderately numerous and large;

(3) Well marked — lesions numerous and large

(4) *Very well marked — lesions very numerous and large*

These authors state that in estimating the degree of nodulation present the whole lung substance has to be considered for the obvious reason that the distribution may be limited or unequal The term small is used for islets of fibrosis with diameters up to 2 millimetres, medium from 2 to 4 millimetres, and large from 5 millimetres up to a centimetre. The term sparse is used to describe the distribution of nodules on the cut surface of the lung of the order of one nodule in a square with a side of 5 centimetres, moderately numerous signifies one nodule per square with a 3 centimetres side, numerous when the square has a side of 2 centimetres and very numerous when the square has a side of less than 2 centimetres.

The nodule must be distinguished from calcified deposits and from the small fibrotic patches of obsolescent tubercu-

losis As noted above, the silicotic nodule in the British cases is usually darkly pigmented, whereas calcified deposits and patches of obsolescent tuberculosis are much less so

In long standing cases the aerated lung tissue between the nodules collapses, thus causing the groups of composite nodules to run together into the large hard, dark pigmented masses already described These masses are commonest in the upper lobes, they may be wedge shaped with the apex of the wedge towards the hilum like deposits of caseous tuberculosis, they are frequently subpleural, and beneath the apical pleura are associated with emphysematous bullae, and eventually they may occupy the greater part or even the whole of the lobe (Figures 1 and 2)

In addition to this characteristic fibrotic nodule the pleura generally shows evidence of old pleurisy in the shape of thickening, usually patchy, with obliteration of the interlobar fissures, and the production of sessile adhesions closing portions or the whole of the pleural sac A layer of soft plastic fibrin, the result of a recent terminal pleurisy is often found at the autopsy, covering the old thickened pleura Pleural effusion is rare

In the lung the most important change apart from the fibrotic nodule, is the presence of emphysema This is so common as to form almost an integral part of the picture It occurs in the shape of bullae beneath the apical pleura, and along the thin anterior margins of the lungs, similar to the emphysematous lesion in other lung diseases But it is seen also in another characteristic form, viz, a compensatory emphysema of the deeper portions of the lung tissue giving rise to an irregular honeycomb appearance

The later stages of the disease are often characterized by a septic bronchitis with muco-pus plugging the bronchi, and a terminal reddening of the lower lobes from congestion



FIGURE 1
Silicotic lung showing massive nodules best marked in upper lobe.

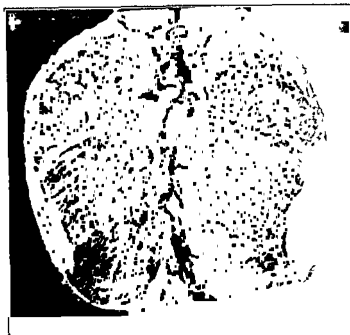


FIGURE 2

Silicotic lung showing extensive nodulation especially in lower lobes

which may go on to a true red hepatization These terminal conditions will be referred to again under the complications and sequelae of the silicotic lung

In uncomplicated cases little of importance may be found in the other organs Myocardial degeneration is not uncommon and is considered by some pathologists to be due to the obstruction in the pulmonary circulation as a result of the fibrosis, but it must be remembered that, as a rule, silicosis as seen in the autopsy room is a disease of the elderly industrial worker whose heart is likely to show signs of wear and tear in

any case Although silicosis is believed to show a biochemical aspect there is as a rule no obvious toxic effect on distant organs in man, such as one finds in gout for instance, though lesions in the kidneys and other organs have been obtained by Gye and Purdy[8] (1922) and others in experimental animals Chronic interstitial nephritis may be found in silicosis but there are generally other reasons for its presence, (e g atheroma) and one cannot with any confidence claim that such lesions are simply the result of a silicotic toxin.

## Microscopical Appearances

*The Silicotic Nodule.* The silicotic nodule is best studied when small and discrete and for this purpose the earlier the nodule is obtained the better. The key to the situation is to be found in the minute aggregations of lymphoid tissue which are normally located, in the lungs of man, in relation to the bronchioles, saccular ducts, small branches of the blood vessels and pleura These aggregations of lymphoid tissue have been carefully described by W.Snow Miller[11] (1937) and should be distinguished from the lymph nodes which are found in intimate relation with the cartilaginous bronchi These small collections of lymphoid tissue are simply minute aggregates of lymph corpuscles without the organised structure of supporting framework and capsule of connective tissue which a true lymph node of the cartilaginous bronchus possesses In relation to the bronchioles they are found lying between them and the accompanying branch of the pulmonary artery. They are also to be seen along the course of the pulmonary artery and the pulmonary vein, in the interlobular septa and beneath the pleura, probably in this case in association with minute radicles of the pulmonary veins but not necessarily so Miller has examined the lungs of elderly persons in order to determine the distribution of this lymphoid tissue in relation to the usual carbon pigment which

accumulates in the lung with age The masses of lymphoid tissue around the bronchioles and the branches of the pulmonary artery were found to be hypertrophied into a common mass, whilst those associated with the pulmonary veins and the venous trunks in the interlobular septa were also hypertrophied He further noted that these masses were usually found where alveoli were in the direct line of the air current against the septa The lymphoid masses beneath the pleura are similarly hypertrophied.

These small masses of lymphoid tissue, therefore, are the sites of the formation of the silicotic nodule, a point which has been well stressed by Strachan and Simson[10] (1930) Pathologists get very few opportunities of examining the nodule in its very early stages in man and the picture tends to be obscured by subsequent events, especially by the presence of pigment, but probably the sequence of events is as follows

Amorphous particles of silica together with carbon pigment reach the saccular ducts and alveoli Here they are taken up by large macrophages, generally designated by the unscientific but expressive term 'dust cells,' and reach the masses of lymphoid tissue described above At this site these 'dust cells' come to rest and are surrounded by fibroblasts In the course of time they degenerate and disappear leaving behind them the insoluble particles of silica and carbon pigment. Meanwhile the fibroblasts have given place to collagenous fibres and one or two minute capillaries may be formed from neighbouring vessels The early nodule is now complete When suitably stained it resembles a small round wicker basket in which the interwoven wicker work may be taken to represent strands of collagenous fibres (Figure 3) In the centre are a few small capillaries, and lying between the fibres are particles of silica and carbon pigment Surrounding the whole, new fibroblasts are formed producing in their turn more collagenous fibres By this process the older fibres in the

## THE COMPLICATIONS AND SEQUELAE OF SILICOSIS

The following are the chief complications and sequelae noted in the post-mortem room.

*Emphysema.* This has already been referred to as an almost constant finding. Occasionally an emphysematous area may become sufficiently large to form a cavity filled with muco-pus and inhaled pigment from neighbouring bronchi Such a cavity may be mistaken for a tuberculous or bronchiectatic cavity but as a rule it does not possess the well defined wall of either of these

*The Heart.* The patient often dies with all the symptoms of cardiac failure. But this is a notoriously unsatisfactory term, and at the autopsy there is usually nothing to be found except a pale, soft, flabby heart muscle with some dilatation of the chambers There are no valvular lesions, and histologically there is little to be made out.

*Septic Bronchitis and Pneumonia.* Secondary infection with one or other of the organisms associated with bronchitis and pneumonia is common, the streptococcus being the organism most frequently found. On section of the lung muco-pus exudes from the bronchi around which are areas of broncho-pneumonia

The combination of a septic broncho-pneumonia and the nodular fibrosis of silicosis may be confused with chronic fibro-caseous tuberculosis, it is never safe to pass over the condition without microscopical investigation.

Histologically the lesions resemble those of septic bronchitis and pneumonia in general, but mention must be made of the hypertrophy of the minute aggregations of lymphoid tissue in the bronchial wall which may be confused with early microscopic lesions of tuberculosis

*Bronchiectasis* Dilatation of bronchi may supervene as a secondary result of the silicotic fibrosis It is not very com-

mon and as a rule does not reach the stage of a fully developed foul smelling purulent bronchiectasis

*Tuberculosis.* Next to septic bronchitis and pneumonia, tuberculosis is the commonest sequela of silicosis Indeed, so common and so intimate a part of the pathological picture is it that some writers have declined to admit that such a disease as simple silicosis exists, and have attributed the whole condition to a chronic form of tuberculosis Silicosis accompanied by tuberculosis is often referred to as 'infective silicosis.' This form of the disease will therefore be treated separately.

*Carcinoma* Bridge and Henry[12] [1928] have proposed that cancer, in order to be classified as of industrial origin, should fulfil the following conditions · (1) The rate of incidence in the occupation under review should exceed that in the general population to a significant extent, and (2) in the occupation concerned there should be sufficient association of the worker with a substance proved experimentally to have carcinogenic properties Suggestions have been made from time to time, notably in the case of the Schneeberg miners in Germany (Rostoski[13] [1928] Schmorl[14] [1928]) and again recently by Dible[15] [1934] in England that carcinoma of the lung is likely to supervene upon pneumoconiosis of long standing The data we possess at the moment however are not sufficient to satisfy Bridge and Henry's postulates and we cannot do more than regard the association of silicosis and carcinoma (and of asbestosis and carcinoma) with suspicion The varieties of cancer found in association with silicosis are those found in intra thoracic malignant neoplasms in general (Gloyne[16] [1930]) They are (1) growths involving the mediastinal glands (2) growths involving the bronchi.

The primary growth involving the mediastinal glands is a rapidly growing white tumour situated in the neighbour

hood of the bifurcation of the trachea and along the course of the two main bronchi. The glands become adherent to each other as they enlarge and coalesce into an irregular collar of growth surrounding the lower end of the trachea, the two main bronchi, and the great vessels. For many years this type of growth was described histologically as a lymphosarcoma, but it is now generally regarded as a carcinoma, an oat cell carcinoma, by reason of its characteristic oval cells, though it is not clear from what part of the respiratory tract it arises. The deep layer of cells of the epithelial lining of the main bronchi has been suggested

The other type of malignant growth is more clearly bronchial in origin. It may arise in large or small bronchi and may be single or multiple It needs to be carefully distinguished from the type of growth described above which has a predilection for the mediastinal glands. These primary bronchial growths extend along the bronchial wall pushing their way through the silicotic tissue as they grow. Ulceration and necrosis with cavity formation is common. This last point is important because the growth cavity may easily be confused with a pyogenic abscess or bronchiectatic cavity. Microscopic sections, therefore, should never be neglected Histologically they are, for the most part, squamous cell carcinomata possessing the usual characteristics with prickle cell arrangement, keratinisation, and cell nest formation

The precise relationship of these tumours to the silicosis in the matter of causation is of course impossible to define This subject will be referred to again under the heading of asbestosis and carcinoma where the pathological relationship has been worked out further.

### Silicosis and Tuberculosis

If, when silicosis is accompanied by tuberculosis, the two pathological processes were always found side by side as they

are in silicosis and pneumonia, there would be little difficulty in differentiating between the two diseases Unfortunately, this is not always so The two are often intimately associated in such a way as to produce what is tantamount to a separate disease In theory, there are three possibilities in the modes of onset and of progress. (1) the tubercle bacillus is already implanted in the lung before the silica exposure begins, the tuberculous focus being either (a) quiescent or (b) active, (2) the tubercle bacillus reaches the site as a secondary infection in a lung which has already become silicotic, the silica exposure (a) having ceased or (b) being still in operation, (3) the tubercle bacillus and the silica particles reach the healthy lung more or less simultaneously This third possibility must be rare The legal mind is very much alive to these possibilities and questions relative to them are frequently asked of medical witnesses in court Unfortunately Nature does not always paint her pathological pictures with an eye to these fine distinctions and we must be careful to distinguish between the post-mortem appearances actually found and the inferences drawn from them as to the mode of onset. For descriptive purposes we may classify the various findings of the post-mortem room into three main groups, though it will be realized that in actual practice the picture is not always so well defined (1) silicosis with obsolescent tuberculosis, (2) silicosis with open tuberculosis, (3) tuberculo-silicosis

*Silicosis with Obsolescent Tuberculosis* Three cardinal signs of obsolescent tuberculosis are generally recognisable (1) dense pleural adhesions, especially in the upper part of the pleural sac tethering the apex of the lung to the apica[l] pleura, (2) fibrotic contraction and scarring of the viscera[l] pleura and subjacent lung tissue without adhesions, agai[n] especially well marked at the apices, (3) calcified nodul[es] In the presence of a chronic fibrotic disease of the lung su[...]

as silicosis these signs are often difficult to interpret. Calcification is probably the most reliable, but the South African pathologists have described small deposits of calcium in the silicotic nodule, and of course, calcification of non-tuberculous origin may take place in a vessel wall or around a foreign body. A calcified nodule beneath the pleura, or attached to the wall of a small bronchus, or in a mediastinal gland generally means obsolescent tuberculosis. If calcified deposits are found in the mesenteric glands, this fact may be taken as corroborative evidence. Generally speaking, however, silicosis found in the presence of obsolescent tuberculosis does not present any unusual features.

*Silicosis with Manifest Tuberculosis* Frank active tuberculosis when it occurs is most commonly a terminal event. The following varieties are to be found. (1) pleural effusion, tuberculous empyema, or pyopneumothorax; (2) old cavities, often multilocular, with fibrotic walls having silicotic nodules adjacent thereto or actually forming part of the wall; (3) rounded foci of caseous tuberculosis with a sharply defined black rim of compressed pigmented lung tissue surrounding the foci; (4) miliary spread from an older tuberculous focus, (5) tuberculous broncho-pneumonia (which cannot always be distinguished from that due to other organisms without microscopic examination). We have here a composite picture resulting from the invasion of the pulmonary tissues by two very different types of invaders. The one is an insoluble particle which reaches the distal parts of the bronchial tree but does not reproduce, the other is a parasite with a capacity for multiplication and for being lysed by the tissues The former remains strictly bronchogenic in its spread, the latter soon reaches the blood stream and is disseminated widely. This applies also to the next group, but to a less marked degree

*Tuberculo-Silicosis.* It is convenient if not essential to have

a special term by means of which to designate a form of silicosis accompanied by tuberculosis in which the one disease is modified in association with the other. The term tuberculo-silicosis is used in this chapter in this sense only

The modification is generally in favour of the tuberculosis, which is frequently of such a chronic fibroid type that it cannot be recognised until histological examination is made

The first and essential feature is the excessive fibrosis The second characteristic is the closely interwoven texture of the two disease processes, tuberculosis and silicosis, in contradistinction to the picture seen in silicosis with manifest tuberculosis The third is the tendency to calcification

Tuberculo-silicotic nodules vary a good deal in appearance as a result of the distribution of pigment, the silicotic portion being as a rule darkly pigmented and the tuberculous portions free from pigment. The result is that at least four different types of nodules may be recognized ( 1 ) a nodule with a soft gray tuberculous centre and a pigmented silicotic periphery ( 2 ) a nodule with a darkly pigmented silicotic centre and a gray tuberculous periphery ( 3 ) a pigmented silicotic centre surrounded by a ring of small gray satellite tubercles ( 4 ) tuberculous and silicotic nodules side by side, the one compressing the other These various types of nodules may result in the affected portions of the lung having a mottled or marbled gray-black appearance ( Figure 5 )

Histologically the tuberculo silicotic nodule has the following characteristics . ( 1 ) a tendency to be more irregular in shape than the simple silicotic discrete nodule, the foci running together more readily into composite nodules ( 2 ) collagenous fibres form only part of the nodule, the rest being caseous material ( 3 ) minute deposits of calcium are not uncommon ( 4 ) giant cells are few and generally lie just outside the nodule ( 5 ) tubercle bacilli are variable, the more caseous looking the nodule the greater is the likelihood of



*Figure 5*

Tuberculo silicotic nodulation, the upper half of the photomicrograph showing caseous nodules, the lower a *tuberculo-silicotic* nodule with a caseating centre (×80).

finding them, they should be searched for in the neighbourhood of the giant cell (6) lastly, small branches of the pulmonary artery may exhibit an obliterative thrombo-arteritis which may be mistaken for silicotic nodules.

Of course, not all nodules found in a case of tuberculo-silicosis exhibit these characteristics; *simple silicotic nodules and uncomplicated fibro caseous tuberculous nodules occur*

Kettle and Archer[17] [1933] have investigated some of the problems encountered in this disease, which are particularly difficult in Great Britain, where a low-grade tuberculosis with excessive fibrosis is common apart from dust hazards. They believe that the differential histological diagnosis depends upon the amount and distribution of collagen

in the tissue reaction This is a matter of degree and they cannot define any delimiting standards. They suggest, however, that a chemical analysis for silica will often help when taken in conjunction with the other findings. In their experience an 'ordinary dried lung' (amongst which may be included uncomplicated tuberculosis) does not contain more than about 0 2 per cent of silica whereas in the tuberculo-silicotic lungs they investigated, analyses up to 2 7 per cent were obtained. It goes without saying that a number of different portions of lung should be selected for analysis (Figure 6)

SOME UNUSUAL PATHOLOGICAL VARIETIES OF SILICOSIS

The range of industries whose workers encounter free silica is a wide one and it is not surprising that certain forms of the disease should, from the pathologist's point of view, appear unusual At least three of these call for special descriptions

*Silicosis in Coal Miners* This condition is occasionally referred to as anthraco-silicosis and is met with in a variety of forms in those parts of the coal field which show a silica risk A great deal has been written on this subject and a distinction is not always made between pure anthracosis and anthracosis with silicosis One of the best recent accounts is that of Cummins and Sladden [18] (1930) dealing with miners in the Welsh coal field *in the 'hard headings' of which silica dust is* present. The outstanding feature of their cases was a lung showing deep black to dark gray consolidated areas on a background of chocolate coloured or gray aerated lung substance The consolidated areas usually varied from small 'maculae' in the lower lobes to large confluent areas in the upper Histologically the characteristic appearances were a diffuse fibrous hyperplasia of the lung with massive dust accumulation in the alveoli and lymphatics together with

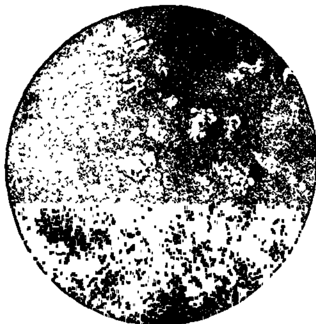


FIGURE 6

Quartz particles from stone-mason's lung. Photographed with dark ground illumination (× 1200). (Courtesy of *Tubercle*)

the occurrence of fibrous nodules, some of which showed little or no lamination while others were markedly laminated or whorled.

*Silicosis in Hematite Miners.* Stewart and Faulds [19] (1934) have described a series of cases in which both hematite and silica dust were present in large amounts in the lungs (sidero-silicosis). The hematite found was a ferric oxide producing a fine dry dust and gave to the affected parts of the lungs a bright red colour. The condition appeared to resemble closely the coal miner's lung in its histology, a diffuse or massive fibrosis with much dust accumulation and a 'few discrete rounded nodules of the usual silicotic type.'

In the one case the fibrotic areas appeared as bright red areas on a dark brown aerated lung background, in the other, the fibrotic areas were almost coal black on a background of chocolate coloured or gray aerated lung Both these types of disease are of much interest to the pathologist and doubtless other similar conditions will in the course of time be described

Broadly speaking there are two large groups of cases (1) one in which the free silica dust is preceded or accompanied by massive accumulation of a fine non siliceous, non toxic dust which acts chiefly in choking up the alveoli and the whole lymphatic drainage system of the lung, (2) a group in which the free silica is accompanied by a silicate which may have toxic and lethal properties of its own (In this connection *the writer is indebted to* Dr E R A Merewether *for a* lung which shows the effects of exposure to free silica and asbestos )

*Acute Silicosis* Of recent years a number of cases of so called 'acute silicosis' have been described, characterized generally by a short exposure to a siliceous dust and a rapidly fatal issue The records have been collected and reviewed by Middleton [20] (1936) who gives the employment history of the largest group so far reported In most instances there has been something unusual in the kind of dust to which the worker has been exposed Often it has been a highly siliceous one mixed with an alkali or powdered soap for the manufacture of abrasive soap powders, and the suggestion has been made that a silica sol may be quickly formed in the presence of alkalis which is responsible for the rapid course of the disease

Descriptions of the pathology are still very meagre The first autopsy record was that of MacDonald [21] (1930) and colleagues. They described the lower lobes of the lung as being solid, greenish-gray in colour somewhat resembling

marble, with innumerable tiny pinhead nodules of a lighter gray colour, whilst the upper lobes were *still aerated* but contained similar nodules Microscopically there were large fibrotic patches which were practically acellular and were *surrounded by small-celled infiltration. They found no histological* evidence of tuberculosis and no tubercle bacilli

Kettle[22] (1932) after examination of material from cases of acute silicosis considered that the sections showed 'acute tuberculosis enhanced, no doubt, by the silica present'

Stuart McDonald[23] (1932) recorded an example in which post-mortem examination revealed minute silicotic nodules in the lung not unlike miliary tubercles and strongly resembling the lesions of experimental silicosis in animals

Chapman[24] (1932) described a case in which the lungs, at autopsy, were about half the normal size, the middle two-thirds being shrunken, fibrotic, and almost as hard as stone. Histologically the fibrotic nodules were found to have hyaline centres

Obviously *the scanty nature of the* pathological material available at present prevents our constructing a satisfactory picture of the pathology of the disease, and there is a danger *of confusing the issue, as Kettle pointed out, with* cases which are primarily acute tuberculosis A distinction should be drawn between (1) workers who have had a very short but intensive exposure to siliceous dust *but whose lungs*, at autopsy, contain large and even massive patches of fibrosis resembling those of the ordinary chronic form of silicosis and (2) those with an equally short exposure but *with miliary* silicotic nodules resembling those of the experimental animal, as in Stuart McDonald's case (The natural life of the laboratory animal is short and therefore more strictly comparable with an acute than a chronic silicosis in man.) It might be best, at the moment, to confine the term acute silicosis to this

second group In any case it is inadvisable to include in the definition cases showing any evidence of tuberculosis, which can play such queer tricks in association with silicosis

## ASBESTOSIS

The word asbestosis was introduced into medical literature by Cooke[25] (1927) and Stuart McDonald[26] (1927) to designate that type of pneumoconiosis induced by the inhalation of asbestos dust Several varieties of the mineral are used in industry, including chrysotile (white) crocidolite (blue) and amosite, the first named being the commonest A good account of the geology of the mineral is given in a monograph by Hall[27] (1930)

### Naked-Eye Appearances

In an uncomplicated case the pathological signs seen at autopsy are chiefly confined to the thoracic cavity, as follows

*Pleurae* Old dense adhesions are common, and are nearly always sessile, producing in the end complete symphysis of the pleura, the long pedunculated bands so frequently seen in tuberculosis are rare Apical adhesions however, occur and the interlobar fissures may be completely closed

In the least affected portions the pleura retains its transparency but as the disease advances it assumes a typical ground glass appearance and eventually exhibiting stiff, yellow and horn-like plaques some 2 to 3 inches in diameter (The older writers on non industrial fibroid lung were conversant with this type of thickening and likened it to cartilage ) At the apices there may be definite scarring, but whether this is to be regarded as an invariable sign of healed tuberculosis is doubtful Here and there, generally near the apices also, the pleura may be thin over emphysematous bullae protruding from the lung Lastly, covering and obscuring these signs of

old pleurisy are seen evidences of recent inflammation in the shape of reddish patches of lymph arranged in thin flat gauze-like layers or as small shaggy processes Occasionally little patches of recent haemorrhage may be seen, such patches becoming browner and darker with age.

Displacement of the mediastinum is unusual

The changes seen in the parietal pleura are similar to those found in the visceral pleura, but less marked and the horn-like plaques are absent.

Pleural effusion is rare, owing to the obliterative character of the disease.

*Lungs.* The asbestos worker is generally a town dweller and therefore the pathological picture, like that of silicosis, is superimposed upon a certain amount of anthracosis which forms a background This confusion between the two is however greater than in silicosis.

The asbestosis lung is generally large and of increased weight, though as the disease progresses it may become contracted from excessive formation of fibrous tissue. It does not collapse when the thorax is opened, and like fibroid lungs in general, is firm, tough, and dense. (Figure 7)

On section blue-black patches of irregularly polygonal shape about 1/8 to 1/4 inches across are seen, they are hard and rough to the touch and are surrounded by a gray framework of thickened interlobular septa. This forms the typical unit of the asbestotic lung, corresponding to the silicotic nodule In the upper lobe these polygonal areas are usually discrete with unaffected lobules lying between them In the lower lobe they tend to run together and become confluent Engorgement of the lung or an actual bronchopneumonia frequently provides a deep red background for the blue-black polygonal areas Much pigment and the blue-black polygonal patches of asbestosis are found in excess in the peripheral portions of the lung immediately beneath the visceral pleura,

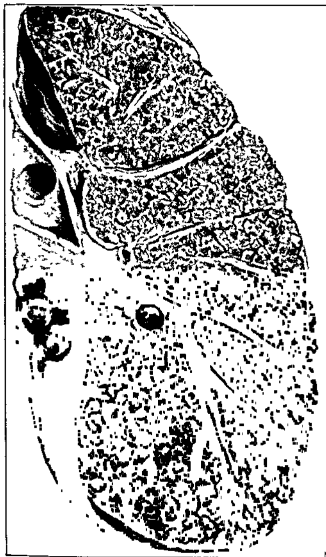


FIGURE 7
The asbestosis lung

exemplifying the 'pleural drift' of the dust so common in pneumoconiosis The interbronchial and other lymph glands of the middle mediastinal group which drain the lung, are intensely pigmented and fibrotic.

The pigmented polygonal areas of asbestosis are roughly coincident in size and shape with pulmonary lobules, and if examined with a hand lens the cut ends of small bronchi, sometimes dilated, can be seen. The fibrotic nature of the lung is better appreciated when the specimen has been fixed. The pulmonary vessels and bronchi now show thickened walls and the cut ends stand widely open, but the writer has not seen a typical extensive purulent bronchiectasis such as is sometimes described in chronic fibroid lung of non-industrial origin.

Occasionally the cut surface of the lung shows a honeycomb appearance due to the breaking down of alveolar structure analogous to the spongy emphysema described by Cummins and Sladden [18] in anthracosis This is commoner in asbestosis than in silicosis Cavitation without tuberculosis, such as is described by Cummins and Sladden in anthracosis, has not been found in these areas

*Mediastinal Glands* The absorption of asbestos fibres into the mediastinal glands, is a much more difficult task for the lung than is the absorption of carbon or even small particles of free silica, but in the course of time the glands become small and hard with a thick fibrous envelope, and trabeculae are seen as white lines traversing them. When bronchopneumonia supervenes, they become secondarily enlarged and reddened.

*Other Organs.* Apart from the complications and sequelae to be dealt with later, there are few naked-eye changes in other organs As in silicosis the heart muscle may be thin, pale, and flabby, and the spleen hard and fibrotic, but there is little else of importance.

MICROSCOPICAL APPEARANCES

*The Asbestos Fibre and the Asbestosis Body*. The asbe
lung is essentially a dusty lung It contains in addition t
asbestos fibres and the bodies formed from them, ca
pigment common to all town dwellers, jagged carbonac
particles such as are present in coal miners' lungs, bl
pigment, and metallic impurities of the asbestos dust wh
tend to accumulate in the neighbourhood of the asbesto
bodies. These various structures are apt to obscure the re
causative factor, the asbestos fibre If asbestos dust be ex
amined with dark-ground illumination, it will be seen t



FIGURE 8
Asbestotic lung showing the respiratory bronchioles packed with asbestosis bodies, fibres and pigment (x 360)



FIGURE 9
Asbestotic lung showing reticular fibrosis of the alveolar walls (× 150).

consist of fibres of varying length mixed up with particulate matter of irregular shape and often of reddish colour, representing various mineral impurities. The fibres themselves are straight and highly refractive, and of course, vary in length according to the thoroughness with which the crude material has been broken up. They may extend across the whole field of the microscope or they may be no longer than an anthrax bacillus They can even be broken up sufficiently small to make a suspension which can be inoculated intravenously into the blood stream of the rabbit without producing emboli In the raw material the fibres are held together in thick, straight bundles which, when crushed, are split up into

smaller and smaller bundles, until eventually very fine fibres are obtained. No branching has been observed. Under dark-ground illumination the fibres appear to be brightly illuminated like the filament in an electric bulb, and when very thin have a bluish luminosity They are readily distinguishable from the irregularly shaped silica particles of the silicosis lung. (Figures 8, 9 and 10)

The asbestos fibre is of a highly resistant nature, hence its use in industry It will withstand heat, and is not destroyed by mineral acids It has been stated that with high-power magnification minute pores can be perceived in the fibre which render it capable of absorbing, to a certain extent, dyes

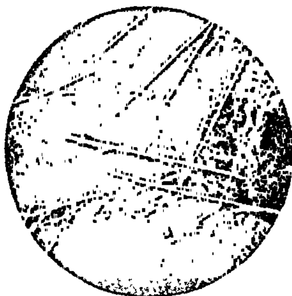


FIGURE 10
Asbestos fibres (crocidolite) Photographed with dark ground illumination (× 1200)

such as methylene blue. The writer has not been able to confirm this.

The fibres are found in large numbers in the lungs Their detection is readily achieved by destroying the surrounding lung tissue with concentrated sulphuric acid, but in ordinary stained sections the presence of these fibres is often obscured by other foreign particles.

In the lung of man two types of tissue reaction take place. The one is the thickening of the fibre, as a result of deposition of iron containing material along its course, to form the asbestosis body. The other is the cellular reaction to the fibre.

The asbestosis body has had a curious history In 1914 Fahr,[28] demonstrating to the Medical Society of Hamburg specimens and photomicrographs of the lungs from a case of pneumoconiosis occurring in an asbestos worker, observed the presence in the lung of a large number of crystals and stated that similar structures had been seen in 1906 by Marchand and Riesel, who had speculated as to whether they were due to the inhalation of asbestos dust or were a haemoglobin derivative No pictures are given in Fahr's communication, but it is probable that what Marchand and Riesel and Fahr saw was the asbestosis body However, these descriptions attracted little attention at the time and seem to have been forgotten.

The next step came in 1927 when Cooke and Hill[29] and Stuart McDonald[30] described certain 'curious bodies' found in the lungs of an asbestos worker who had died in 1924 Cooke had previously recorded the case in 1924 but had not at that time mentioned these bodies Cooke and Hill wrote of them as follows · 'They are yellowish-brown in colour and do not stain with the usual stains, but give the Prussian blue reaction There is a uniformity in appearance, group distribution, and fructating heads that make one think of a fungus The hyphae are verruciform and discoid, and definite spores

are seen.' Pathologists to whom the material was submitted suggested various possibilities, viz., diatomaceae, casts of small cavities, particles of asbestos fibre that had become coated with colloidal matter, fungoid bodies

Later in the same year, Stuart McDonald, to whom post-mortem material had been submitted from the same case by Cooke, gave a fuller description of the bodies He regarded them as foreign bodies associated with the asbestos and was satisfied that they were neither vegetable nor animal in origin, and quoted another case of an asbestos worker in whose lungs he had found similar bodies

The first illustrations were given by Cooke and Hill in their original description Subsequently they were depicted by Simson [30] (1928) in autopsies of South African cases His paper also included a microphotograph of the bodies produced experimentally in the lung of the guinea-pig by Mavrogordato, the first occasion on which the bodies had been so produced.

At this stage Stewart and Haddow [31] (1929) made the suggestion that the bodies should be called 'asbestosis bodies,' a term which has now been generally adopted

Finally Gloyne [32] (1929) showed in the following experiment that the body consisted of an asbestos fibre coated with a substance soluble in a strong acid

A wet preparation of the bodies was placed on the stage of the dark-ground illumination microscope, and the position of a readily recognisable group of these bodies noted Concentrated sulphuric acid was then run very gently under the cover slip, the bodies being kept under observation all the time They began slowly to dissolve, and in the middle of the structure of each appeared a typical asbestos fibre At the end of half an hour practically all the material of which the bodies were composed had disappeared, leaving only faint ghost like outlines with refractile asbestos fibres within them

The diversity in shape of the bodies makes it difficult to write a satisfactory description of them. The following summary comprises the chief points: (1) Marked variation in length (24 to 60 microns) and breadth (12 to 24 microns) Gardner and Cummings[33] record them as long as 250 microns in experimental animals The short forms are sometimes ingested by phagocytes (2) Golden-yellow colour. Gardner and Cummings have pointed out the resemblance in colour to haemosiderin deposits in tissue in which haemorrhage has occurred (3) Homogeneous structure. (4) The central fibre. This can be best seen by reducing the illumination. The *fibre may sometimes be detected projecting beyond the body.* (5) No differentiation with polarised light (Cooke and Stuart McDonald). (6) Tendency to be arranged in irregular clumps and clusters (7) Very early forms unsegmented ('sausage shaped'). (8) Later forms crenated, segmented, or resembling a series of oval discs or beads strung together in necklace form. (9) Bulbous or pointed extremities (or one bulbous and one pointed suggesting 'heads and tails') (10) Generally quite straight, occasionally curved, very rarely S-shaped. When examined in a wet preparation slight pressure on the cover slip will sometimes cause them to bend owing to the elasticity of the central fibre (11) Forms seen 'end-on' look like knobs or door-handles Small secondary knobs and bosses sometimes seen thereon

The bodies do not stain with the ordinary aniline dyes, but according to Gardner and Cummings, Wright's blood stain gives them a 'greenish cast.' They will also retain neutral red slightly, but these partial reactions cannot be called true staining reactions Cooke and Hill and Stuart McDonald, in *their original descriptions, noted* that the bodies could be stained by means of the Prussian blue reaction, and Gloyne has shown that they become black on treatment with ammonium sulphide Haematoxylin, which also has an affinity

for iron, can be made to colour them to some extent, but it is more a deposit of stain than a true staining reaction.

With regard to their formation, no such bodies have been found in crude asbestos or in asbestos dust, and it seems practically certain that they are only produced when the fibres gain access to living tissues and remain there for a considerable period of time.

Gardner and Cummings showed that the golden-yellow material which covers the fibre contained iron, whilst Simson and Strachan put forward the view that the iron-containing substance was of cellular origin

From a study of a large number of different forms of asbestosis bodies, the present writer has endeavoured to build up a complete picture of formation from the original inhaled fibre Figures $11_1$ to $11_3$ show deposition of some material on the fibres to a varying extent When completely covered the body thus built up has a sausage like form without any differentiation from end to end (Figure $11_4$). These thickened forms next begin to show definite fissures (Figures $11_5$ to $11_7$), giving the appearance of incomplete segmentation at irregular intervals In Figure $11_8$ this segmentation has advanced a stage further, the fissures now extending almost to the mid-line of the body and occurring at irregular intervals along its length These fissures produce the appearance of a head or knobs at the ends (Figures $11_9$ and $11_{10}$). As they become deeper and more numerous a completely but irregularly segmented body results (Figure $11_{11}$). When the fissures reach the straight central fibre they appear as (a) a small number of long segments as in Figures $11_{12}$ to $11_{18}$, or (b) a large number of short segments as in Figures $11_{19}$ to $11_{34}$ These short segments may be regular in size and shape resembling beads on a necklace (Figures $11_{32}$ to $11_{34}$), or the segmentation may be complete at one end of the body and incomplete at the other

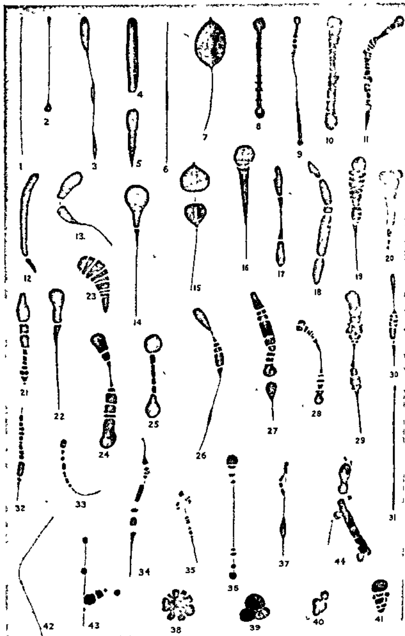



FIGURE 11

Stages of formation of the asbestosis bodies.
( Courtesy of *Tubercle* )

In this final stage some of the segments or even small portions break away leaving bare lengths of fibre between the remaining segments (*Figures* $11_{35}$ to $11_{37}$) Very *occasionally* these broken fragments can be actually seen *in* the process of breaking away This is well shown in Figures $11_{35}$ and $11_{37}$. Bodies viewed end on or obliquely are seen in Figures $11_{38}$ to $11_{41}$.

The asbestosis body would appear, therefore, to be formed from the original fibre by a process of (1) deposition of some material around the fibre so as to thicken it, (2) fissure of this material giving rise to the appearance of segments; and (3) fragmentation or the separating off from the asbestosis body of fractured portions of the deposited material

The central fibre having been satisfactorily demonstrated, the question arises, of what does the golden material consist which forms the segmented coating? Iron certainly enters into its composition, for, as far as we know, all asbestosis bodies give an iron reaction This metal is present in crude asbestos in varying amounts, but it is also present in haemoglobin On the whole, the evidence is in favour of the iron being derived from the tissues — probably from blood pigment — but the point has not been conclusively settled, and it seems probable that the golden yellow substance is the product of a colloidal reaction between the fibre and blood protein

Asbestosis bodies have considerable power of resisting the ferments of the gastro intestinal tract when swallowed with sputum and have been found in the faeces. They also resist heat The effect of acids varies a good deal In sections of lung tissue they resist decalcifying fluid which contains 7½ per cent of nitric acid, for several weeks Concentrated sulphuric acid leaves only a mere semblance of a ghost-like body with a central fibre Hydrochloric acid has the effect of bleaching the bodies, the yellow colour is taken out almost immediately

leaving a colourless skeleton of the body, suggestive of a minute hyaline urinary cast Nitric acid has less effect and glacial acetic acid none. The bodies will resist 20 per cent caustic soda for twenty-four hours without losing their golden-yellow colour. They become black when digested with trypsin

The inference from these reactions would appear to be that the asbestosis body consists of at least two portions surrounding the fibre — viz, an acid resistant material which forms a framework and gives the body the characteristic shape, and a golden-yellow pigment contained therein which is not acid resistant.

The question now arises, what inference can be drawn from the presence of the asbestosis body? Since these structures have not been found in any other disease, their presence is an indication of exposure to asbestos dust, whilst the fact that the bodies have not been found in asbestos dust itself implies that their formation takes place in the body — probably as a result of a colloidal reaction with the blood proteins, in which the iron plays an important part They have been found in the guinea-pig by different observers from seventy days to six months after exposure to dust. Simson has recorded the bodies in a human lung with a history of only two months' exposure.

Lastly, can it be inferred from the presence of the asbestosis body that fibrosis necessarily exists in the lung? The presence of the asbestosis body implies a tissue reaction to the fibre, but it does not necessarily follow that this reaction runs parri passu with the production of pulmonary fibrosis. On one occasion the writer found fine collagenous fibres in the tissues of a guinea-pig as early as twelve days after subcutaneous inoculation of asbestos fibres, but the amount was very minute A heavy dose of fibres may be needed — and even then years may elapse — before pulmonary fibrosis is sufficient to be detected by physical signs or roentgenogram during life. It

would be unsafe, therefore, to accept the presence of the asbestosis body as undeniable evidence of fibrosis, all we can say at present is that the asbestosis body is an expression of a tissue reaction to a foreign body acting as a benign irritant, and that fibrosis of an appreciable extent may or may not be a part of that tissue reaction.

There is some evidence that asbestosis bodies are not formed with the same facility in all the tissues of the body. Thus they have not yet been described in the skin warts to which the workers are liable The writer has examined without success sections of a wart which was known to have been present for many weeks, and has failed also to find them in the skin of a guinea-pig seven months after the asbestos fibres had been introduced into the cutaneous tissues Dewirtz also does not make any mention of asbestosis bodies in sections of warts he examined from the skin of Moscow factory workers Furthermore, even the lung varies in its reaction to asbestos in different animals. Gardner and Cummings[33] (1931) record that the lung of the rabbit apparently offers a much less favourable environment for the production of the bodies than does the lung of man and of the guinea-pig The rat also does not produce bodies readily, whilst Schuster[34] (1931) failed to find them in the lungs of a dog which had been kept on factory premises for the purpose of ratting for several years, though fibrosis of the lungs was present

*The Lungs* The tissue reaction to the asbestos fibre in the lungs depends partly on the non-absorbable character of the particle and partly on its silicate content. There is little leucocytic reaction, the cells chiefly concerned being the lining epithelium of the respiratory bronchioles and the large macrophage which is the striking feature of the cellular reaction in many of the non-pyogenic pulmonary diseases. The bronchial epithelial cells do not phagocyte the asbestos fibre They are simply desquamated into the lumen of the tube as

a mechanical result of the irritant action of the fibre. The macrophages are identical with the so called 'dust cells' of the lung and surround the fibre in an attempt to engulf it. Frequently the fibre is too long to be completely engulfed and looks as though it was sticking into the cell with one end protruding, like a pin in a pincushion. Whether this is in all cases a true statement of facts is not quite certain owing to the liability of the long asbestos fibre to lie obliquely rather than in one plane. (Figure 12)

In addition to these tissue phagocytes, asbestosis giant cells and fibroblasts are seen The former are foreign body giant cells probably resulting from partial degeneration of small collections of the above-described large phagocytes which have



FIGURE 12

Asbestosis bodies (low power) in unstained section of lung (x 520) (Reproduced from article by W Burton Wood and D S Page) (Courtesy of *Tubercle*)

lost all their envelopes In early lesions, e g. in the guinea-pig, the cytoplasm has a pigmented stippled appearance in contradistinction to the almost structureless look of the tuberculosis giant cell. Sometimes the faint outlines of the degenerated individual cells which form this composite 'cell' can be seen The cell also varies considerably in size according to the number of phagocytes composing it Finally, the nuclei of the asbestosis giant cell are large and less numerous than those of the tuberculosis giant cell and have not the same tendency to be arranged round the periphery The fibroblast does not appear to differ in appearance in asbestosis from the fibroblast of other varieties of pneumoconiosis It is found around bronchioles and alveoli, in interlobular septa and in the subpleural connective tissue (Figure 13)

The trachea and main bronchi are little affected in asbestosis The bodies can be seen on the surface of the epithelium, having been carried there by bronchial secretion, and occasionally they may be seen in the mucous or fibrous layer of the wall of the main bronchus surrounded by phagocytes and free pigment The writer has not yet seen them actually in the tissues of the trachea In the intra pulmonary bronchi, from the point where they enter the lung to the point where they terminate in respiratory bronchioles, the most marked change is the desquamation of the epithelium, and lying amongst the desquamated epithelial cells asbestosis bodies, fibres and loose pigment are frequently to be found, often plugging the lumen The basement membrane is thickened and the fibro muscular coat surrounded by a ring of collagenous fibres The elastic fibres generally remain intact, but in some places they are found to be contracted together as though definitely increased and when ulceration takes place they may be found in the sputum Asbestosis bodies may be found in any part of the wall

Gardner and Cummings[33] (1931) have shown by animal

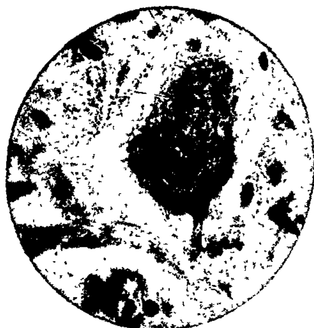


FIGURE 13

The asbestosis giant cell showing phagocyted asbestosis bodies and fibres (x 1200) (Courtesy of *Tubercle*)

inoculation experiments, however, that the chief site of asbestos dust localisation is the respiratory bronchiole. In man, asbestos fibres, pigment, cellular debris, phagocytes and asbestosis bodies — the latter frequently in radially arranged clumps — are all to be found choking the lumina of these tracts, blocking the narrow alveolar ducts, and filling the air sacs and alveoli. Lastly, around these distal portions of the bronchial tree and of the alveoli a fine network of collagenous fibres is formed, ultimately producing a generalised reticular fibrosis which is a distinguishing feature of the disease. When this diffuse fibrosis has reached an advanced stage all alveoli in the area may be compressed out of recognition Whorled nod-

ules such as those of silicosis are rare but are occasionally found in these areas of advanced fibrosis Asbestosis bodies are occasionally present in the proximal parts of the plexus of lymphatics around the smaller bronchi, and become more numerous towards the respiratory bronchioles and are plentiful at the level of alveolar ducts The lymphatics accompanying the branches of the pulmonary artery less frequently show asbestosis bodies or pigment In the small lymph-nodes draining the lung tissue asbestosis bodies as a rule are not plentiful owing to the difficulty the fibres encounter in entering the afferent ducts The aggregations of lymphoid tissue in the walls of the small bronchi are hypertrophied

Changes in the vascular system are relatively unimportant, presumably because the toxicity of asbestos is not marked There may be an increase of collagenous fibres around branches of the pulmonary artery and asbestosis bodies may also be seen lying in the connective tissue between the artery and the wall of the bronchiole The capillaries are of course involved in the reticular fibrosis of the alveoli wall of which they are the chief constituents and may be compressed almost to the point of occlusion The pulmonary venules are also involved in this fibrosis Lynch and Smith (1930) have reported asbestosis bodies in the thrombi of veins

*The Pleura* The surface of the visceral pleura is generally extensively denuded of epithelium, with patches of fibrin containing a few desquamated endothelial cells lying on the denuded surface The subendothelial portion is much thickened by the formation of collagenous fibres with many fibroblasts, and new formed blood-capillaries are numerous, especially in the deeper layers Asbestosis bodies are scanty and tend to be short and stumpy — probably the larger fibres do not penetrate so far — and usually lie surrounded by connective tissue in small groups Fibres are also occasionally seen, singly or in sheafs The elastic layer is generally intact

The interlobular septa may show asbestosis bodies, fibres and pigment with or without collagenous thickening. In two cases whorls of connective tissue resembling discrete silicotic nodules were found by the writer near the point where the septum joined the pleura — which is the point at which the pulmonary deep lymph-vessels in the septum anastomose with the subpleural lymph-vessels

*Histology of other Organs* H.L.Stewart, Butcher and Coleman [35] (1931) have found asbestosis bodies in the spleen The present writer has failed to find them in any of the abdominal organs, even in the walls of the stomach and intestines where swallowed sputum might conceivably carry the bodies.

To summarise, the typical histological lesions of asbestosis are three. (1) The pouring out of large macrophages into the most distal portions of the bronchial tree and of the alveoli to surround and as far as possible phagocyte the invading fibres, (2) the formation of the asbestosis giant cell; (3) a generalised reticular fibrosis surrounding the bronchioles, alveolar ducts, alveoli and air sacs, capillaries and venules, the interlobular septa and pleura, ultimately resulting in a complete obliteration of all lung structure.

## Complications and Sequelae

The chief complications and sequelae of pulmonary asbestosis are as follows: —

(a) Purulent bronchitis.

(b) Bronchopneumonia, which is best seen in the parts of the lung least affected with the asbestosis This point is probably of importance because it explains why bronchopneumonia is such a fatal disease in asbestosis — it puts out of action the last remaining functioning portions of the lung

(c) Pulmonary tuberculosis In a series of 100 cases published by Burton Wood [36] (1934) and the writer, active

tuberculosis was found in 21. The cases coming to autopsy have been of the acute caseous type (in one case with a generalised miliary spread), with few Langhans giant cells and no obvious sign of healing. Asbestos fibres, asbestosis bodies and tubercle bacilli have been found lying side by side in the caseous centres, but there is evidence in animal experiments (Gardner and Cummings) that asbestosis bodies disappear in caseous areas. In the series quoted above there was one case of pyopneumothorax with lardaceous disease following treatment by collapse therapy. A number of cases of healed and obsolescent tuberculosis have also been found at autopsy. These are probably not more numerous than in postmortem work in general, but they indicate that the supervention of asbestosis does not necessarily mean the lighting up of quiescent tuberculosis.

(d) Empyema. In the series mentioned above there was one case of tuberculous empyema.

(e) Bronchiectasis. Dilated bronchi are occasionally met with, but bronchiectasis with fusiform or cavity like dilatations containing foul pus have not been encountered by the writer.

(f) Carcinoma has been found in 6 cases examined by the writer at autopsy. Until more statistics are available it is impossible to draw any very definite conclusions as to the relation of the two diseases. The points noted up to the present are (1) 5 out of the 6 cases of carcinoma have been of the squamous variety, the sixth being an oat cell carcinoma; (2) 4 out of the 6 have been associated with asbestosis which was fairly advanced and of long standing; (3) in 4 cases the carcinoma consisted of an isolated nodule only; (4) the nodules were in the portions of the lung most affected by the asbestosis—i.e. basal or peripheral; (5) 2 only showed secondary growths, one in pericardium, thoracic wall, liver and kidney, the other in thoracic vertebrae; (6) histologi-

cally the squamous carcinoma appeared to advance along the mucosa of the small bronchi, pushing aside the pigment, asbestos fibres and asbestosis bodies as it grew, whilst in the 1 case of oat cell carcinoma observed, the tumour grew outwards from a centre in a compact mass without any tendency to confine itself to prolongation along and infiltration of the bronchial mucosa, (7) death occurred in 4 of the cases whilst the growth was still small (i.e. not more than 1 to 1½ inches in diameter.)

## Clinical Pathology and Post-Mortem Examination

Laboratory investigations of silicosis and asbestosis have been few. The following is a brief summary, and the reader is referred to text books on biochemistry for the more detailed account of silica estimations.

*Sputum* Patients usually produce very little sputum until the late stages of the disease when the material expectorated is often the product of intercurrent bronchitis and, owing to the excess of mucus, may be of small value. The sputum of asbestosis is more likely to yield helpful information than that of silicosis by reason of the asbestosis body.

*Asbestosis* The sputum is generally thick and mucoid — frequently so thick that it resembles semi-coagulated egg albumin. In the latest stages small nummules of pus appear The asbestosis body may occasionally be found by examination of a direct smear, but as a rule a concentration method is necessary for its detection. The one in general use is that originally devised by Stewart and Haddow[31] (1929). It is carried out as follows: — About half an ounce of sputum is dissolved in an equal quantity of undiluted formalin, centrifuged, the deposit washed and examined microscopically. This may be done by drying and fixing a direct smear of the deposit (which need not be stained) or by mounting a small

quantity under a cover glass as in examining a urine deposit Stewart[37] (1934) suggests that the occurrence of asbestosis bodies in clusters is indicative of tissue destruction in the lung sufficient to liberate the clusters from the small bronchioles in which they have been formed

Stewart and Haddow (1929)[31] have also obtained the bodies by direct lung puncture, whilst the present writer has obtained them in faeces in a case in which it was believed that the sputum was being swallowed

*Silicosis.* Here there is no distinctive structure to fall back upon and the detection of particles of free silica by direct illumination and the ordinary microscopic technique is impossible Burke[38] (1935) has described a technique which unfortunately requires a larger amount of sputum for its performance than is available in the great majority of instances A large quantity of sputum (100 cc or more) is gently heated in a beaker until most of the water is evaporated, 50 cc of agua regia is then added and the mixture left at room temperature for twenty-four hours with occasional shaking It is then centrifuged at high speed for one hour, the deposit smeared on chemically clean slides and incinerated to a white or pale grey ash This deposit is examined by polarised light for doubly refractile particles which are generally angular or needle-shaped and vary in size from 1 to 5 microns These particles can be further identified as quartz by mounting in immersion fluids of known refractive indices

*Blood* Schlomka and Nolte[39] (1935) have made blood counts and sedimentation rate estimations on a series of patients with silicosis Their results indicate that this method of approach may be helpful in marking the onset of tuberculosis They found that in various types of silicosis accompanied by tuberculosis the blood sedimentation rate was increased, the total leucocyte count was higher and the propor-

tion of polymorphonuclear cells in the differential count was increased, when compared with the figures obtained in simple silicosis.

King and Stantial[40] (1933) record methods of estimating the silica in the blood by a micro-determination technique of analysis developed from those used by chemists in estimating small quantities of silicic acid in various natural waters Briefly this consists in the reduction of a yellow silicomolybdic acid complex to give a blue colour with aminonaphthol-sulphonic acid, a blue colour being more accurately matched with a colorimeter than a yellow. The methods are too long and complicated for abstraction here and the original papers should be consulted. Both soluble and total silica are examined, the former with 2 cc and the latter with 5 cc blood. The authors state that there appears to be an absence of any insoluble particulate silica and of any organic compound of silicon in the blood so that the figures for soluble and total silica are approximately equal.

*Urine.* King and Stantial[40] have also applied the above methods to the estimation of silica in small quantities of *urine, viz, 5 cc*

Silica is a natural constituent of animal tissues and the deductions which can safely be made in cases of silicosis call for a good deal of experience. King and Dolan[41] (1931) found that silica which was absorbed from the lung or other parts of the body into the blood stream was rapidly excreted in the urine. The elimination of silica in the sputum was higher than in persons having no silica exposure Attempts *made to influence the absorption of silica* from the lungs by the administration of alkali were inconclusive Comparing the blood silica content of non-silicotic and silicotic persons (all of whom had tuberculosis) King and Dolan were unable to detect any significant differences The results obtained by these workers accord with those obtained with numerous

other biochemical substances in disease There appears to be a 'tissue threshold' which enables the body to maintain the constituents of the blood on a fairly even keel until the last stages of disease Biochemical investigations, therefore, in the present stage of our knowledge do not help the clinician very greatly in assessing the functional capacity of the silicotic or asbestotic lung, though they are invaluable in all research work on the subject

*Autopsies.* The following details in the making of post-mortem examinations in these cases have, in the writer's practice, been found worth attention

The lungs should be opened in such a manner that they can be compared with the roentgenogram and the chest diagram of physical signs made by the physician during life. It makes things unnecessarily difficult for those who have to assess compensation claims to find that the lungs have been cut to ribbons in all directions The writer's practice is to place the lungs, with trachea and bronchi attached, on the operating table with their posterior surfaces uppermost and then to make serial horizontal slices, which can be replaced, from the mid-line of the mediastinum outwards to the pleura If this operation can be postponed until the lungs have been hardened in formalin, so much the better

The natural tendency of the pathologist is to reserve the most diseased portion of the lung for histological examination Whilst this is in general sound practice, the portion thus selected is often so altered by fibrosis or tuberculosis that satisfactory study is difficult The portion of lung which, naked eye, looks least affected by the disease is often the best for microscopy Here the silicotic nodules may be found lying in the midst of relatively healthy tissue and can be examined readily In cases of doubt a portion of interbronchial lymph gland should also be selected, if there are silicotic nodules anywhere in the specimen they are most likely to be found

here. A minimum of three sections should therefore be chosen, (1) the most typical portion of advanced disease; (2) the healthiest looking piece of lung, and (3) an interbronchial lymph gland. If possible (1) and (2) should include pleura, whilst portions of lung representing intermediate stages are also selected as required in difficult cases. The writer uses a chest diagram similar to those employed by physicians and marks thereon the position of all portions of lung selected for histological study.

*Histology*. In silicosis where the demonstration of the whorled nodule is all important, a good stain for the collagenous fibre is essential. No doubt pathologists will differ on this point, but the writer has a strong preference for Van Gieson's stain. Reliance on a simple routine haematoxylin stain is liable to lead one astray, especially in tuberculo-silicosis. The only safe rule is to stain three sections from each block of tissue with (1) haematoxylin and a good counter stain for cellular structure, (2) a collagenous fibre stain such as Van Gieson or Mallory and (3) Ziehl Neelson for tubercle bacilli.

These rules apply with less force to asbestosis. A warning may be given, however, with regard to the resistance properties of the asbestosis body.

In studying the microscopical picture of the different organs one error must be guarded against, viz., the assumption that the asbestosis body has been actually formed at the site in which it is found. Although these bodies probably do not easily migrate far, if at all, during life from the position in which they are formed, they are easily moved in dead tissues by various manipulations. Moreover, they are highly resistant to cleansing, and glassware should be carefully cleansed by concentrated sulphuric acid. In forming an opinion as to whether an asbestosis body has been actually formed where it is found lying in the tissues the following points are help-

ful: (a) the presence of amorphous brown pigment, phagocytes and fibrous tissue around, (b) its relation to surrounding structures (bronchiole, capillary, etc.) The cardinal point in the histology is the liability of the asbestos fibre to be held up at the distal end of the respiratory bronchiole.

Silica particles cannot be detected satisfactorily in ordinary sections mounted in Canada balsam and viewed with transmitted light. With a mounting fluid of high specific gravity such as bromobenzene, particles are more readily detected, but it is only by the use of crossed nicols in a petrological microscope that silica particles can be demonstrated with any certainty. Viewed in this way the quartz particles stand out clearly on a dark background For the actual technique of the petrological microscope the reader is referred to special mineralogical textbooks.

Irwin[42] (1934) has devised a simple method of micro-incineration which enables the observer to detect silica particles in situ in silica nodules in sections After removal of the paraffin from the section (which should not be more than 5 microns thick) in the usual way, the slide is heated to 550–600°C After cooling the incinerated section is mounted in concentrated hydrochloric acid covered with a coverslip and examined by direct illumination for quartz particles A small furnace is needed for the incineration The microscope slides vary a good deal in their resistance to the necessary heat and in the writer's experience there are a good many casualties in the way of cracked slides The ash pattern obtained with silicotic nodules by this method is characteristic A somewhat similar technique has been used by Schutz and Braun (quoted by Siegmund and Koppenhofer 1935)

Dark ground illumination also has its uses It may be employed with advantage to detect the characteristic asbestos fibres in wet or dried fibres of sputum and in sections Siegmund and Koppenhofer have also used it in silicosis (quoting

it as the method of Timms) but the present writer has found so many pitfalls that he has discontinued it.

*Extraction of Asbestos and Silica Particles from Lung.* The extraction of asbestos fibres and asbestosis bodies from the cut surface of the lung is usually a simple matter. A direct smear on to a slide or a film from the centrifuged deposit of a little tissue fluid which has been expressed from the organ is usually sufficient. In the old stone-mason's lung, also, quartz particles can sometimes be obtained readily by removing a subpleural nodule with the point of a scalpel and compressing it between two slides.

But in most instances silica particles are much more difficult to isolate. The best technique is that devised by Jones[3] (1933) for the isolation of sericite fibres and termed by him 'sliming'. Portions of the lung freed from preservative by preliminary washing are treated by gradual disintegration with strong nitric acid over a period of several days. The slime thus obtained is diluted about ten times with hot water and the mixture filtered through No. 51 Whatman's filter paper (this is important, since from the ash of many filter papers minute fibres of cellulose which may cause confusion under the petrological microscope are detached). After repeated washings the residue is carefully dried (the fumes given off are inflammable) and the material ignited in the usual way. The light ash is removed with a mixture of bromoform and benzene in a Sollas separator and the minerals which sink to the bottom are washed with benzene, then alcohol, dried, and mounted on slides in Canada balsam. A petrological microscope is necessary for the identification of particles.

*Chemical Analysis of the Lung.* This is done by chemical assay of the wet or dry ash of the lung. In both cases it is necessary to remove the organic matter first. Jones recommends the method of wet ashing (as described above for the examination of silica particles) on the ground that a dry ash

may contain a high percentage of constituents such as phosphoric oxide, sodium oxide, etc, which are removed by his wet ash technique The majority of analyses have, however, been carried out by dry ashing This consists in mincing up large slices of lung, drying at 100° until friable, powdering in a coffee mill, mixing and drying again at 110°, ashing a small portion (e g 10 gms) in crucible, weighing ash and assaying it (e g by gravimetric method of fusion with alkaline carbonates, precipitation of silica by repeated treatment with hydrochloric acid, ashing the precipitate and determining the true silica content of this ash by treatment with hydrofluoric acid) (Kettle and Archer [17] [1933])

King and Stantial [49] (1933) have devised a micro determination technique by means of which small quantities of tissue can be analysed (about 1 gram fresh tissue or 0 25 grams pulverised dried tissue) as in the case of blood and urine mentioned above The advantages of using such a method are obvious in that they afford an opportunity of comparing areas of diseased lung by chemical and histological methods which the ashing of a large slice of lung does not

No hard and fast line can be drawn between the percentage of silica in the dried lung in silicotic and non-silicotic persons, but probably it would be safe to put the upper limit for the non silicotic lung at about 0 2 per cent Silicotic lungs often yield analyses of 1 to 2 per cent

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## V. EXPERIMENTAL PATHOLOGY

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### EXPERIMENTAL PNEUMOCONIOSES

IT WAS once maintained that silicosis, the best known form of pneumoconiosis, could not be studied by experimental methods for it was a chronic disease which took years to evolve and was commonly complicated by the most chronic manifestations of an infection like tuberculosis However, experience has demonstrated the fallacy of this contention With pure silica it has been possible not only to reproduce tissue changes that simulate the lesions of human silicosis in several short-lived species but to demonstrate that such changes alter native susceptibility to tuberculous infection It has been necessary to curtail the essential factor of time but this has been largely compensated by exaggerating the reciprocal factor of dosage While this necessary adjustment may have produced some distortion of the resultant lesions so that they are not always identical with those in human silicosis, the same kinds of cells participate in the reaction and the type of tissue change is the same. ( Figure 1, *a* & *b* ) All of these effects are specific and cannot be produced with non-siliceous materials On the other hand, the mixed dusts, com posed of silica and other minerals, which apparently cause silicosis in different industries have not uniformly excited comparable reactions in animals In searching for an explanation for his apparent failures, the experimentor is beginning to discover principles which may be much more important than his successful reproduction of silicosis with pure silica It would now appear that the non-siliceous components of a mixed dust are not merely inert diluents of the silica but



FIGURE 1

*a* Vertical section lungs of white rat Inhaled pure chalcedony 9 months, then normal atmosphere 5 months *b* Detail of above Note marked swelling of collagen.

that they exert specific effects upon the silica either in the atmosphere or after it has been inhaled Evidence is accumulating from various sources to indicate that some types of mineral particles may accelerate the action of silica on the tissues ; others may retard it or perhaps prevent its inhalation from the atmosphere. The conditions governing such modifications have not been defined but the experimental methods now developed offer a means of attack Certainly a silicosis that develops with unusual rapidity should be capable of experimental reproduction The limitations imposed by the span of life in small animals may preclude a complete evaluation of inhibitory effects Nevertheless experiments should point out principles which govern reaction to various combinations of dusts and they may disclose the manner in which silica injures the tissues

Other forms of pneumoconiosis have not been studied so thoroughly. The lack of significant degrees of reaction to nonsiliceous dusts has been sufficiently well established to assert with a reasonable degree of certainty that the pneumoconioses of this class are essentially pigmentations with little or no effect upon respiratory function Finally, there is the group of conditions caused by silicates, which Badham designated as 'silicatoses' Only one of these, asbestosis, which may be produced by several of the fibrous silicates, is generally recognized as a disease Its early manifestations have been reproduced in animals but the evolution of its more advanced phases still awaits demonstration The evidence of other forms of silicatosis is largely roentgenographic. Experimental studies with this group of minerals have thus far yielded nothing to support a belief in their capacity to provoke progressive fibrosis

The basic facts that have already been discovered in an intensive study of man in various dusty environments and the reliable methods of procedure now in possession of the

experimentor offer hope that in time he may be able to answer the perplexing questions that still enshroud the reaction to all kinds of inhaled dusts.

With these general considerations in mind the achievements of the experimental pathologist can now be examined Much of his work remains to be done; some of it will have to be repeated because of the discovery of certain pitfalls of which he was not aware in his earlier efforts But many of his observations are undoubtedly sound and can be used as the basis for further investigation.

## Experimental Methods

The experimentalist has a variety of methods at his command. Obviously the most satisfactory way to study the effect of *inhaled dust* is to make animals inhale it This procedure is expensive, time-consuming and requires very special equipment but it constitutes the only means by which the effect upon the lungs of atmospheric suspensions of dusts or mixtures of dusts can be determined. It permits investigation of the physical properties of such dusts as they exist in the atmosphere. It allows the protective mechanisms of the upper respiratory tract to come into play and to admit or exclude the different components of a dust from the lungs It permits the foreign particles to accumulate slowly and without gross trauma to the tissues and it does not interfere with the natural mechanisms of elimination. In fact the inhalation method is the only one to demonstrate decisively whether a given kind of dust suspended in the atmosphere will injure the lungs

All other methods merely indicate whether particles of a certain composition are capable of producing reaction in the tissues. They are most useful in eliminating the substances which are not irritating and which consequently do not need further investigation. Based upon the *injection* of aqueous

or saline suspensions of particles, they have the advantage of being easy to perform, of requiring no special apparatus and of producing results in relatively short periods of time. All of them are subject to the one fundamental objection that they test the action of an aqueous rather than an air-borne suspension of particles and it is now known that the properties of such suspensions are not necessarily identical With proper precautions this objection may not be particularly serious as long as pure substances are being tested In dealing with mixtures of harmful and benign minerals exclusive reliance upon injection techniques may lead to false conclusions Many of the proposed procedures are also unsatisfactory because such overwhelming doses of the substance under investigation are injected that confusing non-specific inflammatory reactions are produced This is particularly true when the particles are introduced directly into the organ where their effects are to be observed In most instances the test organ is not the lung but this is probably of no moment for experience has demonstrated that free silica and all non siliceous minerals *in particulate form* have the same effect upon connective tissues regardless of the organ in which they are located Apparent exceptions are the *fibrous* silicates known collectively as asbestos, which seem to exert their specific action only in the lungs In spite of these objections, injection methods have their place and the ones which are technically satisfactory have contributed materially to the solution of the pneumoconiosis problem

One injection test, (Gardner and Cummings' 1933) [1] in the writer's opinion obviates the non specific effects that have been mentioned and results in localization of particles by physiological mechanisms It is based upon repeated intravenous injections of small fractional doses of particles suspended in physiologic salt solution Full grown rabbits are injected by ear vein with one gram of particles, 1 to 3

microns in diameter, administered in 20 doses over a period of sixty days.* Particles introduced by this method are retained in the capillary systems of various organs long enough to be phagocytosed. Most of those of the standard size adopted for this test pass through the vessels of the lung and come to rest in the sinuses of the spleen and the sinusoids of the liver. A few lodge in the capillaries of the bone marrow but practically none remain in the renal vessels. The mobile phagocytes then transport them into the connective tissues where they either remain in masses or enter the lymphatics and are carried to regional lymphoid tissues. Wherever a sufficient number of particles is thus collected one expects to discover evidence of the reaction characteristic of the material. If uncombined silica has been injected a progressive fibrosis occurs which becomes visible to the naked eye within three months and continues to progress throughout the life of the animal (Figure 2, *a*, *b*, *c*, *d*, *e*, *f*.) All silicates and all non-siliceous particles that have thus far been tested have provoked such slight amounts of chronic inflammation within

* A sufficient quantity of particles between 1 and 5 microns in diameter is prepared by elutriation in water or other suitable fluid. They are washed

gradually worked into a uniform suspension, adding more drops of the saline solution as necessary. It may require several hours to work up the full gram of particles but when finished one has a thin watery paste with no clumps and all of the particles wetted. For routine purposes enough salt solution is added to bring the final suspension up to 1 per cent strength. Suspensions of relatively insoluble dust are again sterilized in an autoclave. Tests with pure quartz have demonstrated that such treatment does not increase its irritating properties. More soluble dusts have to be sterilized dry and suspended immediately before injection. Injection of 5 cc each are made every three days into the ear veins of full grown rabbits until the full dose of 1 gram has been introduced. To protect the ear veins from

the last injection and the others six, twelve and twenty four months after

periods of one and two years that microscopic sections must be used to detect it. (Figure 3, *a*, *b*, *c*, *d*, *e*)

Many other injection methods are being used with considerable success in spite of their objectionable features For example, the intraperitoneal technique of Sayers and Miller[3] (1931) will give gross evidence of reaction to free silica within a few weeks or months The procedure is simple and requires no special apparatus One or two cc of a 10 per cent saline suspension of dust particles that will pass a 350 mesh screen are injected directly into the abdominal cavity of a guinea pig. Particles slowly soluble in water provoke a transitory reaction which later disappears, leaving no trace of the dust or the cellular changes. Full doses of more soluble substances like sodium silicate and dolomite are apt to kill the animal immediately because of their strong alkalinity, but smaller quantities may be tolerated. Insoluble dusts produce a minimal amount of inflammation which does not increase with the elapse of time and the injected particles remain in the tissues for an indefinite period Free silica either alone or in association with other materials causes progressive proliferation resulting in the formation of fibrous nodules several centimeters in diameter after six to eight months. The silicates produce nothing like the reaction to free silica, some of the hydrous forms are capable of causing necrosis or death of the tissues but such reaction is of microscopic proportions and it has not been followed by significant amounts of fibrosis

On the basis of their experience with this test Miller and Sayers classified the activity of dusts as proliferative, absorptive or inert The only cause of proliferative reaction which they or other investigators have discovered is free silica The slowly soluble compounds of calcium are generally classified as absorptive. The inert minerals include most of the silicates and a wide range of insoluble non-siliceous substances It has



FIGURE 2 See caption on page 265.

been assumed that non-proliferative reactions in all animals killed within a period of one year after intraperitoneal injection indicates that a mineral is incapable of causing significant responses by living tissues Perhaps this assumption is unwarranted, longer contacts might result in an interaction between foreign particles and the body fluids that would provide a stimulus to cell proliferation But even if time is the essential factor, which has hitherto been too greatly curtailed, one would expect some evidence of a progressive change within a period of twelve months Such has not been the case in an investigation of some 30 different minerals, other than free silica, investigated in the writer's laboratory The tissue changes have remained stationary or actually retrogressed.

Another method of testing is that of intratracheal injection of particles suspended in physiologic salt solution It was used extensively by Kettle[3] (1930) and by Lemon[4] (1933) The injection of fluid into the air spaces causes a transitory, inflammatory edema of the lungs which may be followed by more chronic changes in the presence of irritating silica. Because of the difficulty in differentiating between

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FIGURE 2

Intravenous injections of pure free silica *a* Normal quartz, one year after completing injections Replacement of most of parenchyma by diffuse and nodular fibrosis *b* Normal quartz 22 months after completing injections Higher magnification of nodular and diffuse fibrosis with necrosis still in progress along cluster of parenchyma cells at upper border *c* Tridymite 80 days after completing injections Destruction of most of parenchyma cells and replacement by diffuse fibrosis *d* Tripoli one year after completing injections Extensive nodular fibrosis, large area of diffuse fibrosis at lower border *e* Opal, one year after completing injections Residual fibrosis and collections of large phagocytes in portal connective tissue Reaction subsiding rather than progressing *f* Diatomite, two years after completing injections Widespread nodular fibrosis replacing much of parenchyma



FIGURE 3 See caption on page 267.

the non-specific effects and the specific ones produced by the particles themselves, the lungs are not as good a place to study the results of such injections as the lymph nodes which receive their drainage. Particles accumulate in the tracheo-bronchial nodes by physiologic mechanisms and there produce specific reactions commensurate with their physico-chemical composition.

As already indicated, results can be obtained by direct injection of suspended particles into the subcutaneous tissue, the testis, the eye and other organs, but in all cases the non-specific reaction is confusing. A series of intratesticular injections now under observation demonstrates more evidence of cellular reaction to many of the silicates than to non-siliceous substances. Since this observation is at variance with the findings in intravenous and intraperitoneal tests with the same substances it is difficult to evaluate the results.

Intracutaneous injection was tried and for a time it appeared that this might prove a valuable diagnostic procedure. Injection of 0.1 cc of a 0.2 per cent suspension of silica particles under 3 microns in diameter *into* the skin of a guinea pig will cause a local inflammation in a few hours. Between eighteen and forty-eight hours thereafter a central

FIGURE 3

Rabbit livers after intravenous injection. *a* Aluminum oxide, 3 months after completing injections. Particles collected in large phagocytes situated in portal connective tissues. No fibrosis. *b* arnet, 6 months after injections. Phagocytes filled with particles ttered widely through sinusoids and in portal areas. No fibrosis. *c* Feldspar, one year after injections. Large phagocytes ed with particles in portal areas and in sinusoids. No fibrous ibrous sericite, 8 months after injection. Giant phagocytes still ered through sinusoids. No fibrosis. *e* Talc particles, 2 years injection. Giant phagocytes collecting in and about a portal ; some lymphocytic infiltration. Focus of proliferation about ocytes in parenchyma with early necrosis and slight polynu-infiltration. No fibrosis.

zone of ulceration develops so that the area resembles a strongly positive tuberculin reaction. Comparable injection of non-siliceous particles causes only a transitory swelling and redness which quickly subsides leaving a central spot of pigment if the particles are colored. Unfortunately some of the silicate dusts also produce rapid inflammation and it has not always been possible to differentiate them from reactions to free silica.

A wide choice of *animal species* is open to the experimentor and he can select one adapted to his purpose by reason of anatomic peculiarities White rats of strains that are resistant to pulmonary infection make excellent subjects for dust inhalation experiments as their lungs develop nodular fibrosis during a six to eight month exposure to pure silica These lesions more nearly resemble the silicotic nodules of human beings than any yet observed in other animals (Figure I *a, b*) But the white rat is naturally resistant to tuberculous infection and little is known of the cause of its immunity. Guinea pigs, on the other hand, develop a modified type of silicotic nodulation at a somewhat slower rate; the reaction is specific for silica but it bears less resemblance to the human lesions (See Figure 5-*c*) However, guinea pigs are susceptible to tuberculosis and so much is known about their response to this infection that they are invaluable agents Rabbits of certain breeds react readily to inhaled silica and develop nodular fibrosis. Such nodules tend to undergo necrosis with subsequent calcification, a change encountered in certain human beings who are apparently free from infection The rabbit's large, superficial ear veins makes it the animal of choice for intravenous injections (Figures 2 *a* to 2 *f*) This species is extremely susceptible to tubercle bacilli of bovine type but less so to those of human origin In rabbits alterations of native susceptibility induced by coexistent silicosis have been well demonstrated

by infection with human type tubercle bacilli. Cats react well to inhaled silica; their relatively small hearts and large lung fields make them admirable subjects in which to follow the course of a pneumoconiosis in roentgenograms. Dogs react rather slowly to silica injected by vein, but advanced though somewhat localized visceral changes in the liver have now been produced. Domestic fowl develop nodular fibrosis of both the lungs and the appended air sacs when exposed to the inhalation of silica (Figure 5 a). The specificity of such reaction in chickens has not been tested by parallel exposures to other dusts but there is no reason to doubt that this species is unique. Even gold fish and tadpoles develop a characteristic necrosis followed by fibrous proliferation when suspensions of silica are injected into their tissues. White mice react in a similar manner to injected silica but they apparently have very effective upper respiratory protective mechanisms for after a year's exposure by inhalation only a few phagocytes filled with silica are observed in their lungs.

## General Tissue Responses to Various Kinds of Mineral Particles

*Non-siliceous Particles.* Tests of this nature in different animal species are demonstrating that all relatively insoluble non siliceous particles, except those generally recognized as poisonous, tend to produce the same cellular changes. The particles are ingested by mononuclear phagocytes which transport many of them into the connective tissues. There they remain or through the agency of the lymphatic vessels they are carried to the nearest lymphoid tissue. The same phagocyte seems to be capable of taking in an extremely large number of particles of the size under consideration. Its cytoplasm enlarges to accommodate the burden and in many instances its nucleus divides to form a small giant cell. In the latter case the newly formed nuclei tend to arrange them-

selves in a single row beneath the surface membrane Overloaded cells are apparently paralyzed for they generally remain at the site where they develop. The smaller cells which happen to have ingested fewer particles exhibit their customary amoeboid activity and they are chiefly responsible for the accumulation of particles in the stroma and lymph nodes. In the case of non-siliceous substances they may pile up enough particles to produce gross pigmentation but this is associated with the most insignificant changes in the fixed tissues. There may be small accumulations of lymphoid cells and perhaps a proliferation of a few fibroblasts. Once formed such reaction does not progress or at least no progression has been observed over a two year period. While the histologist might be justified in describing this picture as a cellular fibrosis it is far too slight to be detected by gross inspection or palpation

The manifestations of such reaction in the livers of rabbits injected intravenously consist of isolated mononuclear and giant phagocytes scattered through the sinusoids and collected in small masses in the connective tissue about the bile ducts. (Figure 3, *a, b, c, d, e.*) A few lymphocytes may infiltrate the latter area The picture is essentially the same whether observed immediately after completing the injections or two years later The spleens of such animals usually show more phagocytes and larger giant cells, most of which remain in the sinuses and cause little or no reaction in the fixed tissues.

The result is the same whether the foreign body is hard and insoluble (e g. diamond, aluminum oxide, silicon, the carbide of silicon, rutile) or softer (e g. coal, iron oxide) or slowly soluble (e.g gypsum, marble, calcite, limestone). With the latter substances the cells tend to disappear as the particles dissolve Still more readily soluble materials like dolomite are toxic because of the rapid change in hydrogen

ion concentration which they produce, but such effects are of no interest in this connection

*The silicates* constitute a group of minerals whose biologic activity needs thorough investigation for they have frequently been suspected of causing pulmonary fibrosis in industry If the generally accepted solubility hypothesis is valid many of them should be capable of such action for they are dissolved or otherwise affected by water The recent literature contains several papers by petrologists who warn of the potential hazards from silicates These authors have argued that since the asbestos silicates can cause pulmonary fibrosis, other forms that are also affected by water should produce similar reaction They assert that asbestosis, and by inference "silicatosis" in general is merely a silicosis produced by the silica left after the body fluids have leeched the bases out of the silicate molecule The validity of their speculations awaits demonstration

The roentgenograms of persons exposed by occupation to the dusts of silicates like feldspar, mica and talc offer the most obvious and at the same time the most puzzling evidence of the existence of corresponding silicatoses Many of these films reveal localized massive shadows which should probably be interpreted as a result of infection but there are some in which a fine, uniformly distributed reticulation characterizes the picture Whether such shadows are actually due to reaction to the particular silicate with which the individuals last worked, should be determined by careful study of post-mortem material. Chemical and petrographic analyses must be made to determine whether there may not be appreciable quantities of free silica as well as silicates in the tissues. Histologic study of sections must demonstrate the character and distribution of the lesions, both those due to dust and those arising from associated infection The possibility that certain kinds of dust particles in excessive quan-

tities may of themselves cast shadows on the film must be explored further This idea was once entertained and discarded but Enzer[5] (1937) has now demonstrated that the apparent nodulation of electric arc welders is probably due to aggregates of very fine mineral particles in the lungs with no associated fibrosis. His observation necessitates further investigation of the subject to determine whether other dusts may not have similar effects. Proof of such properties would account for many of the shadows seen in the roentgenograms of silicate workers and would be more in keeping with the experimental observations to be reported in the following section.

To test the capacity of every known silicate to produce reaction in animals would involve a tremendous amount of labor and would serve no useful purpose for many of these minerals are of no industrial importance. The group of 24 silicates on page 273 has been chosen to include varieties used in industry and ones representative of important mineralogic classes. (Figures 3 *a*–3 *e*)

The specimens for this study were supplied by Professor Esper S. Larsen of the Department of Geology at Harvard University Either he or Dr. Alton Gabriel of the U S. Bureau of Mines analysed them petrographically and their chemical composition was determined at the author's laboratory The rocks were pulverized in an iron mortar and then ground in steel ball mills until all of the particles were 3 microns or less in diameter Wherever possible the grinding was done dry but in some instances it was necessary to add water or petroleum ether. To make certain that the composition of the different minerals had not been altered during this treatment the particles were again analysed in various ways Petrographic examination so frequently failed with material of this size that this procedure was abandoned but chemical analysis was always done. A few minerals which

*Anhydrous Silicates*

| *Class* | *Group* | *Variety* |
|---|---|---|
| Polysilicates ($R_2Si_2O_5$) | Feldspar | * Soda microline |
| Metasilicates ($RSiO_3$) | Pyroxene | Rhodonite |
| | Amphibole | * Anthophyllite (fibrous) |
| | " | † Tremolite |
| | " | † Soda tremolite |
| | " | † Amphibole |
| Orthosilicates ($R_2SiO_4$) | " | † Crocidolite (blue asbestos) |
| | Garnet | Beryl |
| | Topaz | Almandite ? |
| | | Sillimanite |

*Hydrous Silicates*

| | | |
|---|---|---|
| Water of Crystallization | Zeolite Division | * Analcite (artificial) |
| Essential Water | Mica Division | Muscovite |
| " | " | Biotite |
| " | " | * Sericite (fibrous) |
| " | " | * Sericite (platey) |
| | Serpentine and Talc Division | † Amosite (fibrous) |
| | " " | † Serpentine |
| Water Variable | " " | † Chrysotile (fibrous) |
| | " " | † Talc |
| | Kaolin Division | * Glauconite |
| | | Dickite |
| | | Halloysite |
| | | Montmorillonite |
| Anhydrous | *Titano-Silicate* | |
| | | Titanite |

* Action observed for a year or more
† " " four months

were appreciably altered were excluded from the study on this account One substance, chrysotile asbestos, was also checked by roentgen-ray diffraction because of a peculiar change which suggested that its composition might have changed When suspended in water, the ground fibres developed a marked tendency to agglutinate into compact ball-

like masses. Dr. William F. Bale of the University of Rochester very kindly compared the roentgen-ray diffraction patterns of untreated and treated asbestos and reported that there had been no change. It was therefore concluded that the treatment with water had not altered the molecular structure. This test was not used for the other minerals.

Suspensions of all of these silicates in physiologic salt solution were injected intravenously into rabbits and intraperitoneally into guinea pigs in the doses already mentioned. The study has not been completed but as noted in the above table the effects of 8 of the minerals have been followed for a year or more and 8 others, for at least four months. Most of the remaining ones have been injected but only a few animals of each series have been killed.

Within the period of observation no rabbit successfully injected with any of these silicates has shown evidence of connective tissue proliferation. Intraperitoneal tests on guinea pigs which concentrate much larger quantities of particles in localized areas have confirmed these observations Two of the silicates have thus far proved difficult to inject intravenously. Glauconite particles agglutinate rapidly in aqueous suspensions and tend to cause thrombi in the rabbits' ear veins. None of the animals could be given more than 0.4 gm of this substance for after 9 injections all of the larger ear veins had been thrombosed. Chrysotile asbestos killed 8 rabbits either immediately or within twenty four hours, again probably because of its tendency to agglutinate in water. Although no thrombi could be found in the cerebral or other vessels it is believed that its action was mechanical rather than chemical because serpentine, which has the same chemical composition was tolerated readily. Furthermore, intraperitoneal injection of chrysotile caused no untoward effects Incidentally the latter has failed to cause either fibrosis or the formation of asbestosis bodies in guinea pigs. Miller and

Sayers (1934)[2] had the same experience on injecting chrysotile intraperitoneally and were forced to classify asbestos as an 'inert' mineral. It is still possible that fibrosis may develop later for none appeared until the sixteenth month in guinea pigs inhaling this substance However, this outcome is not anticipated in view of the excessive doses administered by the injections under discussion.

While evidence of fibrosis has invariably been lacking it cannot be maintained that all of the silicates have been absolutely inert. A rabbit has just been killed two years after intravenous injection of feldspar. The particles have localized to produce pigmentation of sufficient intensity to be visible grossly as a network of fine, yellow lines about each lobule Microscopically the picture is essentially the same as the one at twelve months, shown in (Figure 3–c) The only change has been a further concentration of phagocytes in the portal areas Another rabbit has died of an acute broncho-pneumonia approximately two years after a similar injection of talc particles. In this case the cellular reaction is much more advanced than that at the last sampling period About the portal collections of phagocytes there is a well marked infiltration with lymphocytes The collections of mononuclear cells in the sinusoids show active proliferation and in many of them there is necrosis with an infiltration of polynuclear leucocytes (See Figure 3–*e*) The possibility that some of the reaction in the liver is secondary to the acute infection in the lungs cannot be excluded

Another unusual finding has occurred in animals injected with fibrous sericite The sample selected by the petrographers as the most likely counterpart of the variety that Jones[6] (1933) described as the cause of silicosis has had an effect upon the phagocytes which is unique among silicates Like free silica in the spleen, it has produced epithelioid changes in the monocytes and caused the formation of

giant cells of the Langhan's rather than the simple foreign body type. No other silicate has acted in this manner; the flat scaly sericite and the feldspar produced cells that were indistinguishable from those called out by inert non-siliceous minerals. The exact significance of this cellular change is not understood but there is nothing to indicate yet that it is a preliminary step in the formation of fibrosis as Drinker, Field and Drinker[7] (1934) suggested The last rabbit injected with *fibrous* sericite died of a chronic abscess of the right lung after twenty-one months. The capsule of the dome of the liver immediately beneath the abscess was thick and fibrous but in the other portions of this organ and in the spleen and lymph nodes the reaction was essentially the same as that in the preceeding members of the series If this *silicate were capable of causing fibrosis such reaction* would be expected in the presence of chronic infection

One other effect has been observed which might be of some significance. Anthophyllite, both types of sericite, amosite, chrysotile and glauconite have caused a limited amount of necrosis when injected intraperitoneally into guinea pigs. Such reaction has always involved only small numbers of cells in the centres of masses of phagocytes but it has occurred in all animals injected with these substances. It has persisted without change for at least a year and has never been replaced by calcification In no instance has there been any suggestion of fibrosis at its margin. It is interesting to note that three of these substances have a fibrous structure and that all but anthophyllite belong to the hydrous group of silicates Such changes may be entirely non-specific but the fact that they occurred in every animal in each series and under conditions where non-specific pressure effects are minimal makes it necessary to mention them

Intratesticular injections of garnet, chrysotile asbestos, talc and feldspar have caused inflammatory reaction whose sever-

ity has increased in the order named but for reasons stated above the significance of such findings is doubtful Possibly the more marked responses to talc and feldspar may be indicative of specific irritating properties but if this is the case the effects are only produced by excessive local concentrations

So much space would not have been given to reporting incomplete observations had not Professors Briscoe[9] and Brammall[8] (1937) recently postulated that those silicates which are affected by water should be capable of producing pulmonary fibrosis Since their observations on the behavior of silicate rocks in nature and their test tube experiments have attracted considerable attention it has seemed worth while to present as much biologic evidence on the subject as is now available Thus far their predictions have not been verified by the injection tests While it is still possible that sericite and some of the other silicates may cause fibrosis after longer periods of contact with the tissues this outcome is not yet predictable. As will be shown presently, free silica injected under the same conditions produces its effects within a few months According to hypothesis, the silicates that are more soluble should cause reaction in a shorter period of time. It might be claimed that the whole experimental program is invalidated by the failure to produce fibrosis with asbestos, but as already indicated *fibrous* foreign bodies present unique problems in the lungs which do not exist in other organs However, final conclusions are reserved until after completion of the study

Various forms of *free silica* have been subjected to the same methods of testing This phase of the study is more nearly completed, although certain important types have still to be investigated The following varieties have been under observation for at least a year after injection into both rabbits and guinea pigs

| Crystalline Silica | Cryptocrystalline Silica | Amorphous Silica |
|---|---|---|
| Normal Quartz | Chalcedony | Colloidal Silicic Acid |
| Tridymite (25% cristobalite) | Tripoli (Seneca, Mo.) | Silica Gel (16% $H_2O$) |
| Cristobalite | | Opal (4.1% $H_2O$) |
| Vitreous Silica | | Diatomite (7.6% $H_2O$) |

The three conversion forms of quartz, tridymite, cristobalite and vitreous silica were prepared synthetically by Dr. L. H. Milligan of the Norton Company. It should be noted that the tridymite is not pure but contains about 25 per cent of cristobalite. The amorphous silica in the gel phase is a commercial product of high purity containing 0.033 per cent of $Fe_2O_3$ and 0.00005 per cent MnO as contaminants. It was made from sodium silicate. By petrographic analysis the unground diatomite was found to contain about 10 per cent of quartz but practically all of it was removed by elutriation of the ground material in water. All of the other substances were pure materials.

All of the crystalline and cryptocrystalline varieties of free silica have provoked a progressive fibrosis in both rabbits and guinea pigs. Intravenous injection into rabbits causes a silicotic cirrhosis of the liver and spleen with replacement of more and more functional tissue by hyaline fibrosis. Coincident regeneration of liver cells is sufficiently active to maintain hepatic function and life. One year after completing the injection of 1 gram of 1 to 3 micron particles whole lobes of the liver are reduced to exceedingly hard, white leathery scar, while regeneration has produced irregular lobules of new glandular tissue at the periphery. The spleen becomes large, pale, smooth and exceedingly hard. Microscopic sections may reveal no trace of normal structure. Both in this organ and in the liver there is a diffuse fibrosis in which are embedded characteristic silicotic nodules of laminated hya-

line fibres. Many of the latter have necrotic or calcified centers The only normal elements left in the areas of diffuse reaction in the liver are bile ducts and blood vessels. (Figures 2 a & 2 b)

The evolution of this process has been described in a previous publication. (Gardner and Cummings 1933) [1] Here it suffices to relate that the silica particles coming to rest in the smaller blood spaces of the liver and spleen are engulfed by mononuclear phagocytes Many of these primary phagocytes are killed and ruptured. Mavrogordato's 'ghost cells' are rare in organs other than the lung and only in that of the white rat has the author found them at all common As they break down, the phagocytes release their particles and new cells quickly wander into the area to take up the material thus liberated. Repetition of this process results in an excess of cells Now the number of particles per cell is no longer so great that the phagocytes are killed, they are merely stimulated. As manifestations of such stimulation one sees alterations in the morphology and the physiologic activity of the phagocytes. In the spleen they undergo the same 'epithelioid' changes that occur in tubercle formation and produce giant cells of the Langhan's type. These modifications have never been observed with any other mineral except fibrous sericite. As a further evidence of irritation the stimulated cells move with unusual rapidity They collect in compact nodules in the organ first involved and they migrate quite rapidly into the nearest lymph node Such action concentrates irritating silica in contact with the connective tissues These elements are in their turn affected and undergo primary necrosis followed by proliferation In the liver, the parenchymatous cells are killed with an invasion of polynuclear leucocytes. The destroyed areas are quickly replaced by proliferating connective tissue. A diffuse obliterating fibrosis results from the silica which remains scattered, nodular fi-

brosis develops about collections of silica brought to the portal areas by stimulated phagocytes.

The picture that has been described has been produced repeatedly by injecting 1 gram of silica particles, 1 to 3 microns in diameter. A further investigation of the effect of dosage has demonstrated several points of interest. As little as 0.1 gram will cause nearly as much reaction but in twice the time. Even 0 05 gram produces microscopic reactions but gross evidences of disease are only barely apparent after two years. When the dose is reduced to 0 01 gram the liver develops no lesion but the hepatic lymph node, which receives the drainage of this organ is completely destroyed and replaced by silicotic fibrosis. The lymphatics are capable of completely eliminating such small amounts of irritant so that the viscus escapes. The spleen, on the other hand, has no extravisceral lymphoid tissue. Most of the particles which lodge in its sinuses remain there and as a consequence even the minimal dose provokes a certain amount of nodular fibrosis.

Intravenous injection of too great a quantity of particulate silica is toxic and causes a reaction simulating that from colloidal silicic acid Rabbits will not tolerate more than 0 05 gram of silica in 1 per cent suspension at a single time If one attempts to introduce 0 1 gram the animals die very promptly with acute symptoms of intoxication. They shiver, gasp, run about in an excited manner and fall dead within a few minutes Autopsy reveals an intense engorgement of the splanchnic vessels and perhaps petechial hemorrhages into the serous membranes. In guinea pigs a single dose of 0 02 gram injected into the heart generally has the same effect The age of the suspension does not influence its activity ; fresh preparations are just as potent as ones which have been kept in the refrigerator for several months Comparable suspensions of non-siliceous particles have no such effect. It is therefore

inferred that in sterilizing the suspensions enough solution has occurred to liberate silica molecules, part of which assume a colloidal form

The size of the silica particle has a marked influence upon the rate of reaction All of the results thus far discussed have been produced by injecting particles 1 to 3 microns in diameter. Larger ones cause less active responses by the tissues, smaller ones are more irritating Large splinters of quartz embedded in the subcutaneous tissue excite no more cellular response than pieces of carborundum of the same size Particles of quartz 10 to 12 microns in diameter can be injected into rabbits' ear veins but owing to their size most of them are caught in the capillaries of the lung instead of passing through these vessels to the liver and spleen However, they react just as well in the lung They produce, not a progressive fibrosis, but merely a foreign body tubercle, which changes very little in two years' time Intravenous injection of particles of silica less than 1 micron in diameter causes rapid death from acute degeneration of the liver cells This has been proved for normal quartz, tridymite, cristobalite and vitreous silica The animals have generally died before a half a gram or even less could be injected

Recently a commercial vitreous silica composed of particles too small to be resolved under the highest magnifications, was submitted for a test of its toxicity By indirect methods the particle size had been estimated at 0 0002 microns. It was of course anticipated that this material would prove extremely toxic when injected intravenously for a suspension in water should approximate the colloidal silicic acid used by Gye and Purdy 1 per cent suspensions by weight were freshly prepared before each injection The rabbits tolerated the material without symptoms all of them remained well and were killed at intervals during a year Only the last animal showed gross evidence of disease which con

sisted of small localized areas of fibrosis in the liver Micro scopically there were slight traces of silicotic reaction in all other members of the series. Apparently the particles were so extremely small that most of them passed through the various capillary beds and were excreted in the urine. Colorimetric analyses of the latter in two animals yield high values, 27 17 and 32.95 mg per 100 cc. Only a very few particles remained in the organs to produce disease after a year's time.

Guinea pigs injected intraperitoneally with 10 per cent suspensions of the same forms of free silica particles develop hard white nodules, 2 to 3 cm. in diameter in the omentum and other subperitoneal tissues The centres of these lesions are at first soft and necrotic but after a year many have calcified. The retro-sternal and hepatic lymph nodes are enlarged and show varying grades of silicotic reaction. The mesenteric nodes ordinarily escape.

Aside from the experience with this unusual material just described the experiments have demonstrated conclusively that the intensity of tissue reaction to silica is inversely proportional to the size of the particles The amount of surface exposed to the action of the fluids and cells is the obvious explanation A gram of particles less than 1 micron in diameter presents infinitely more surface than the same weight of 10 to 12 micron particles.

While all of the forms of crystalline and cryptocrystalline silica mentioned above produce reactions of this type there are appreciable variations in the rate and severity with which they act The tridymite has been outstanding. (Figure 2–c) It is extremely irritating and has killed most of the animals prematurely In two different series of rabbits the maximum period of survival was eighty days and many animals died before the injections were completed All showed an acute hepatitis with an associated replacement fibrosis, dif

fuse rather than nodular in type The guinea pigs tested with this substance have also suffered an excessive mortality although repeated attempts finally produced one animal which survived to be killed after eight months At autopsy its peritoneal cavity showed extremely large fibrous nodules with calcified centers Cristobalite was not as irritating but caused a more rapid reaction than quartz, the fibrosis was always diffuse rather than nodular in type Next in order are probably the cryptocrystalline varieties, chalcedony and tripoli, (Figure 2–*d*) although in some of the tests they caused no more reaction than quartz Vitreous silica is about as potent as normal quartz. In the order of the intensity of reaction produced, these different silicas might be arranged as follows *tridymite, cristobalite, chalcedony and tripoli, normal* quartz and vitreous silica

The *amorphous silicas* have yielded surprising results Diatomite (Figure 2–*f*) caused as much and as rapidly progressive, nodular fibrosis as chalcedony While this substance may have been contaminated with a minute amount of quartz, it seems hardly likely that this was the cause of the reaction for other mixtures containing much more quartz have not had a similar effect Amorphous silica (See Figure 2–*e*) as opal excited much less reaction, the nodular lesions remained cellular in type and only a limited amount of diffuse fibrosis developed Two years after completing the injections there was definitely less reaction than after one year Amorphous silica prepared artificially by powdering the gel precipitated from colloidal silica was no more potent than fibrous sericite. It produced epithelioid changes in the monocytes and giant cells of the Langhan's type but no fibrosis developed in one year's time The action of colloidal silicic acid in the dispersed phase was thoroughly investigated by Gye and Purdy [10] (1922, 1924) These authors described in detail the toxic effects upon the liver, spleen and kidney of

rabbits An illustration in their paper shows stimulated endothelial cells and the formation of giant cells With repeated doses of small amounts of colloidal silica they produced a diffuse fibrosis of the liver and spleen.

The results obtained with these different forms of amorphous silica are perplexing and difficult to explain on any basis yet proposed Perhaps no attempt should be made to explain them until more observations have been made. A study is contemplated with a series of silicic acid gels which contain varying amounts of combined water. Possibly this may yield information of value but it seems hardly probable for the active silicic acid in dispersed colloidal form contains the same amount of water as the inactive substance in the gel phase Perhaps the capacity to take up more water may be of more significance If the very marked response obtained with diatomite can be accepted at face value any explanation based upon water content falls. Mr. E. W. Thompson[11] (1937) in commenting upon this finding has suggested that the unexpected activity might be due to the tremendous surface created by the presence of countless, minute holes perforating the shells of the diatomes of which this mineral is composed But if this were true it would seem as if at least *some reaction should have occurred from the silica gel*

The possibility of *"aging" of silica* with consequent loss of irritating properties has received some credence recently. Professor Brammall adopted Heffernan's[12] suggestion (1935) that freshly fractured particles may be more active because of unsatisfied oxygen atoms which are free to combine with bases in the body fluids. This theory assumes that blasting or grinding actually disrupts the silica molecules, an effect which as yet awaits proof. In the aging process it is assumed that the oxygen atoms on the surface of the fractured silica combine with water or with bases and are thus rendered incapable of reacting with the tissues On the basis of this hy-

pothesis Denny, Robson and Irwin [13] (1937) exposed rabbits to silica actually fractured in the dusting chamber by grinding quartz pebbles in quartz lined mills Their animals developed cellular silicotic nodules of the lungs after an exposure of only six months The writer has never tried this type of experiment. With the commercially ground silica used in his laboratory a minimum of eighteen months has been required to produce the same results The rate of development of the lesions was essentially the same whether the exposures were continued for the full period or whether the animals were exposed for six months and then set aside in a normal atmosphere for a period of one year But one cannot be too certain that these apparent differences are altogether due to the character of the silica dust. The daily exposure of sixteen hours was twice that used by the author in his own inhalation experiments with rabbits Furthermore it is not too certain that the concentration of atmospheric dust employed by Denny, Robson and Irwin may not have been partially responsible for the rapidity of reaction While they report konimeter counts of 4000 to 8000 particles per cubic foot of air, it is not altogether justifiable to conclude that these figures are equivalent to 112,000,000 and 224,000,000 particles per cubic foot of air, the results obtained by mathematical conversion Experience is indicating that each type of dust counting instrument has its own standard and that the results obtained with one of them are not convertible into counts or weights secured by other methods This would seem to be particularly true where high concentrations are involved It is felt that judgment on the effect of freshly fractured silica should be reserved until animals can be exposed under comparable conditions to the same dust before and after it has been allowed to age

While it is granted that freshly fractured silica may be more potent than that which has aged for some time the

writer is positive that the aging process is not a progressive one. For his dust inhalation experiments he has been in the habit of purchasing both chalcedony and normal quartz in lots of two tons from commercial sources These materials are received in 200 pound paper sacks and stored in a reasonably dry building but subject to marked variations in atmospheric humidity. The last supply purchased in 1932 has been used for many different experiments started from time to time during the subsequent years. As far as can be determined silicosis has developed just as rapidly in animals which began to inhale these two dusts in 1936 as it did in others exposed four years previously Likewise sterile suspensions of silica particles in physiological salt solution, which are kept in the refrigerator for many months have neither increased nor decreased in capacity to excite reaction in the tissues. These observations indicate that if a loss of potency occurs it is not complete and that silica remains active even when suspended in dilute aqueous sodium chloride solution

## Mixtures of Free Silica and Other Minerals

The reaction to mixtures of free silica and silicate or nonsiliceous particles is not governed solely by the amount of the first substance. Often the adulterating components are not mere inert diluents which reduce the amount of free silica available to react upon the tissues Some of them seem to accelerate and others definitely retard its action. Briscoe[9] (1937) and his co workers have shown that the addition of a variety of substances to suspensions of silica in the test tube alters its solubility They have inferred that the rate of reaction in the body would be correspondingly altered. Denny, Robson and Irwin,[13] (1937) after making similar observations, used metallic aluminum to prevent the development of silicosis in rabbits MacDonald, Piggot and Gilder[14] (1930) and later others have reported cases of rapidly de-

veloping silicosis from the inhalation of mixtures of alkalies and silica in the manufacture of soap powders. McCord, Flemming, Ainslee and Johnston[15] (1936) have published experiments on guinea pigs injected intraperitoneally with mixtures of silica and alkalis which seem to show an accelerated silicotic reaction. No phase of the problem has been investigated sufficiently to permit generalizations but the following experiences are illustrative of inhibitory action Others will be cited later when the effects of inhaled dust are discussed.

Before describing the reactions to injections of mixed dusts it should be mentioned that unusual care must be exercised in their preparation to prevent alteration in composition. This is likely to happen for the hard quartz components will obviously resist grinding longer than softer substances like iron or certain silicates As a consequence the number of fine free silica particles will be comparatively small unless the grinding is continued for prolonged periods Elutriation in water also alters the composition of the mixed dusts owing to variations in settling rates of different components The only satisfactory method of treating mixtures is to grind them until none of the particles exceed the desired maximum size Chemical and petrographic analyses of the material before and after grinding are essential to make certain that there has been no alteration in the different minerals or their percentage composition in the mixtures

Four different mixtures have been injected into rabbits and guinea pigs and the reactions have been followed for a sufficient period to allow an evaluation of their potentialities, 5 others are under observation. Crude *fluorite ore* containing 57 16 per cent of $CaF_2$, 31 06 per cent $CaCO_3$ and 10 46 per cent $SiO_2$ caused a delayed and a typical form of silicotic reaction when tested by the guinea pig and rabbit methods already described Guinea pigs injected intraperitoneally

showed nothing suggestive of silicotic fibrosis in a period of three hundred days. No specific silica reactions appeared in the rabbits until the seventh month when the giant cells in the spleen began to assume characteristics of the Langhan's type. In the one year animal there were microscopic foci of diffuse fibrosis in the portal septa and an early silicotic fibrosis with calcification in the hepatic lymph nodes The last rabbit of the series was killed at the fifteenth month. The fibrosis in the liver had advanced to a stage where it was visible to the naked eye. Microscopic sections revealed a more extensive diffuse fibrosis without nodule formation and without the hyaline swelling of collagen seen in typical silicosis. There was no appreciable necrosis of adjacent liver cells The hepatic lymph nodes were almost completely replaced by hyaline silicotic foci, many of which had necrotic or calcified centers. A control series of rabbits treated with almost pure fluorite particles ( $CaF_2$ 98.7 per cent, $SiO_2$ 0.08 per cent ) developed no significant changes in the same period of time

This experiment demonstrated that the injection of 0.146 gm. of quartz in combination with 0 88 gm. of calcium fluoride and calcium carbonate would cause fibrosis but that the development of this reaction was delayed and assumed a typical form only in lymph nodes draining the liver where lymphatic activity had concentrated an unusual number of silica particles. As already noted (p. 280) one half this amount of silica ( 0 05 gm ) not associated with other substances is sufficient to produce much more fibrosis in only a year's time One can only conclude that the calcium fluoride or the calcium carbonate or both had slowed and modified the effect of the quartz. The particles were in the tissues for they could be seen inside of giant cells The latter were situated in the same locations that they always assume. But the silica did not affect the cells for a long time. Incidentally

both of the adulterants are substances which have been suggested as accelerators of the action of silica. Nicol[16] (1933) and later McNally[17] (1935) postulated that fluorides were responsible for solution of silica in the body. Calcium carbonate is one of the alkalis which McCord[15] (1936) found would accelerate the reaction to silica. The observations just reported are at variance with their findings.

*Granite* from Barre, Vermont is a natural mixture containing about 33 per cent quartz and 66 per cent of the silicates mica and feldspar. It has always been slow to cause silicosis in human beings, exposures of fifteen to twenty or more years being frequently reported. Inhalation by guinea pigs and rabbits has never produced nodular fibrosis within the usual period of a year or two regardless of the concentration employed. (Figures 4 *a* & 4 *b*) To learn more about its action 4 groups of rabbits were injected intravenously with varying doses of granite particles. The first group received 3.33 grams containing 1 gram of quartz, the second 1.67 grams or 0.5 grams of quartz, the third 1 gram containing 0.3 grams of quartz and the last 0.5 gram or 0.15 gram of quartz. All members of the first group, injected with a full gram of quartz, were killed during a period of twenty two and a half months. None of them developed gross evidence of fibrosis although there was enough pigmentation in the portal connective tissue to demarcate the lobules. In an animal killed at the end of twelve months the spleen contained giant cells of the Langhan's type and the liver exhibited occasional minute foci of monocytic proliferation in addition to localized collections of large dust-filled giant cells. (Figure 4 *d*) For the most part, however, the picture resembled that produced by the inert silicates. In the last animal of the series, killed after twenty two and a half months, the microscopic foci of proliferation were appreciably larger, the parenchyma cells adjacent to them were beginning to degenerate and polynuclear




FIGURE 4 See caption on page 291.

leucocytes had invaded them and the centre of the monocytic nodules. (Figure 4 *e*) Even at this time the evidences of activity were localized and in most places the reaction had remained static. Unfortunately there were no survivors in this group so that it was not possible to determine the time necessary to produce progressive disease.

Two members of the second group injected with a dose of granite containing 0 5 gram of quartz were not autopsied until the twenty seventh month when one of them died with gross evidence of coccidiosis Microscopic examination of this animal's liver revealed an advanced cirrhosis with hyaline fibrous nodules and necrosis of the surrounding liver cells In its spleen, however, the reaction was still in the giant cell stage with no evidence of fibrosis The companion animal, with no infection, presented a picture of moderate cellular activity comparable to that produced by twice the dose after twelve months contact with the tissues

The members of the other two groups injected with doses

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FIGURE 4

Inhalation of various dusts *a* Chalcedony, 9 months exposure plus 4 months in a normal atmosphere Domestic Fowl Nodules

and gypsum, 25 months plus 2 months in a normal atmosphere Guinea pig Large dust-filled plaques inside of thick walled air spaces The nodule in the centre along the lower border is cel-

exposure plus 24 months in normal atmosphere Guinea pig Higher magnification of a lesion resembling that caused by hematite (figure 4e) Slight thickening of alveolar walls adjacent to dust filled plaques inside air spaces

of granite containing 0.3 and 0.15 grams of granite respectively were all killed during a period of eighteen months. All but one revealed an inert type of reaction. The exceptional animal, injected with the larger dose and surviving for eighteen months had developed microscopic evidences of fibrosis in the portal areas.

Intraperitoneal injection of granite dust into guinea pigs has failed to produce any evidence of fibrosis within one year's time.

This experiment points to a very definite inhibition of the action of quartz in the presence of the silicates mica and feldspar. It shows that a full gram of quartz in such combination causes only an early type of silicotic reaction in twenty two and a half months, an effect which has been observed with the same amount of quartz alone within twelve days after completing the injections. That *time* rather than the number of quartz particles is the more important factor is indicated by the findings in the group injected with the dose containing 0.5 gram of quartz. Here twenty seven months contact with the tissues caused as much or more fibrosis. While in one such animal there was a complicating parasitic infestation which should theoretically accelerate reaction, in another the result is clearly defined. The significance of the time factor is substantiated by the absence of fibrosis one year after the injection of a large dose of granite into the peritoneal cavity of guinea pigs.

*Ferruginous chert* from the Lake Superior iron mining district has been the subject of intensive study in connection with clinical and engineering investigations in this industry. In many of the mines the rock runs about 50 percent of silica of the chalcedony type.

Intravenous injection of the standard dose of 1 gram of such chert produced a modified form of silicosis which developed less rapidly than the reaction to an equivalent

amount of pure silica The fibrosis was confined to the portal areas and after a year's time it appeared as if an equilibrium had been reached as necrosis of liver cells was no longer visible. (Figure 4 f) In a series of rabbits injected with 2 grams of chert containing approximately 1 gram of silica an advanced stage of sidero-silicosis had developed within eight months, at eighteen months the lesions were more extensive and still progressing The involvement was never as generalized as in animals injected with pure silica, but the affected areas were quite completely replaced by fibrous tissue Intraperitoneal injections into guinea pigs caused large silicotic nodules in a year Apparently hematite in aqueous suspension with silica does not retard the action of the latter to the same degree as the silicates feldspar and mica in granite

*Dust* collected from an air filter located in one of the *Johannesburg gold mines* has been studied because of the general interest in the disease in this industry The sample submitted was composed of small particles but they varied so much in shape and size that they were reground with water in a porcelain mill until none larger than 1 microns in diameter was left Examined under polarized light an excessive number of brilliantly refractile acicular particles was seen Dr. Gabriel furnished the following estimate of the petrographic composition "Essentially quartz and fibrous sericite with platy muscovite, a trace of feldspar and some opaque material (ilumenite?) as accessory minerals"

A partial chemical analysis showed 55 per cent of free silica and 1 18 per cent of combined silica.

Intravenous injection of a suspension of the 1 micron particles of this dust produced silicotic fibrosis but at a slower rate than either pure silica or chert After six months the tissues were only beginning to exhibit cellular activity After sixteen months however there was gross evidence of

fibrosis of moderate severity in the rabbit's spleen and liver. Intraperitoneal injections into guinea pigs caused a reaction of the silicotic type which had not yet become fibrous in eight months The findings with this material are of interest in view of the high content of sericite in the mixture which should, according to the Jones theory, accelerate the reaction to quartz If anything this sample of South African mine dust has caused a slower reaction than might be anticipated.

Other mixtures of silica with coal, carbon and iron in varying proportions are under investigation but their action has been followed for only six months, a period too short to expect any silicosis to develop Their presence in a suspension of fine silica particles prevents the development of acute symptoms on intravenous injection. The action of gypsum with silica dust will be discussed in the following section

### Inhalation of Various Dusts

The action of inhaled dusts is described in a separate section because of its direct practical bearing on the problem of pneumoconiosis There are many methods of subjecting animals to dust inhalation but the types of apparatus need not be described here. The main objective is the maintenance of a rather constant atmospheric concentration of dust in an enclosed space, large enough to accommodate a very considerable number of animals and sufficiently well ventilated so that the humidity does not become excessive Since inhalation experiments must be continued for at least twelve to eighteen months it is essential to use only healthy animals and to take every precaution against the development of epizoötic pneumonia and other infections. The maintenance of proper temperature and humidity and the avoidance of overwhelming dust concentrations are therefore important.

Some method of determining and recording the concentration of dust in the atmosphere is essential for comparative

studies. The writer has employed the Greenburg-Smith impinger technique [18] (1932) since this is the standard procedure in the United States. In addition to making counts at the recommended magnification of 100 with light field illumination, comparative counts are also made under dark field illumination at the same magnification. The latter is quite essential to visualize the excess of small particles less than 1 micron in diameter, which have been employed. (Particles much larger than 3 microns are not used because they are unlikely to produce much result in the limited period of such experiments.) Counts are made at the breathing zone of the animals and large amounts of air-borne dust are collected over periods of hours for chemical analysis. Both are necessary to determine the amount and the composition of the dust actually inhaled. As will be shown, samples taken in various parts of the dusting chamber may have quite a different composition. The counts have been repeated every month or two but the chemical analysis were not rechecked unless the number of particles varied greatly. According to custom the counts are recorded in number of particles per cubic foot. While it is possible to convert this figure to numbers of particles per cubic centimeter, the usual standard in the British Empire, it is doubtful whether the conversion means a great deal. In dealing with the high concentrations used in the experiments to be discussed the different instruments ordinarily employed do not measure absolute quantities. As mentioned above, there is urgent need that some international standard be adopted so that the conditions defining exposures in different countries can be more accurately compared.

The experimental results on which this discussion is based were produced by exposing animals in groups of 100 to 150 to high concentrations of various dusts, composed of particles less than 3 microns in diameter, for periods of one to two or

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more years The average light field dust counts have varied in different experiments from 100,000,000 to 800,000,000 or more per cubic foot, the corresponding dark field counts being 3 to 5 times as high These concentrations have been maintained for eight hours a day, six days a week. The animals have remained in the same rooms for the other sixteen hours of each day and since considerable dust collects on their bedding it is constantly being stirred as they move about. Dark field counts taken at night, after the motor agitating the dust is turned off, usually reveal from 30,000,000 to 80,000,000 fine particles in the air inside their cages. It is therefore obvious that the exposures have been severe; for eight hours daily the dust concentrations have been very heavy but no greater than the worst encountered in industry. In addition there have always been significant amounts of dust in the air throughout the life of the animals

The results of these inhalation experiments performed upon guinea pigs, white rats, rabbits, cats, fowl and a few *individuals of other species have thrown considerable light* upon the evolution of various forms of pneumoconiosis. These can best be illustrated by considering the reactions to 4 different types of dust (1) non-siliceous dust; (2) free silica, (3) mixtures of free silica and other substances; and (4) the fibrous silicate, asbestos. Other silicates have not been tested in this manner owing to the fact that they have caused so little reaction by injection.

By no means all of the particles that are inhaled reach the lungs, some probably possess properties such as wettability and a tendency to clump which favor retention in the upper respiratory tract Since this feature has not been investigated it will be dismissed with the statement that perhaps 50 per cent of the inhaled particles are caught on the sticky mucous membranes lining the tortuous passages of the nose, throat, trachea and bronchi. Ciliary action and

the cough and other reflexes carry them to points from which they can be expectorated. It is logically assumed that mouth breathers inhale more particles into the lungs than persons who breathe through the nose Lehmann[19] (1936) demonstrated that the nasal filter was less efficient in subjects with silicosis than in ones without this disease Unpublished experiments by A J. Vorwald,[20] working in the writer s laboratory, would seem to prove the reverse although the interpretation of his results is difficult He produced a partial nasal stenosis in guinea pigs by cauterizing the nostrils with silver nitrate Most of the animals could not accommodate themselves to breathing by mouth They swallowed air and died with greatly distended stomachs. A few were more tractable and after a period of training they were exposed to high concentrations of hematite dust for about a year When killed both visual examination and chemical analysis showed much less iron in their lungs than in the controls breathing normally. The meaning of this experiment is not clear, but it is mentioned to indicate the difficulties involved in attacking this aspect of the problem.

*Non-siliceous dusts.* The general treatment of inhaled particulate dust which enters the lungs is perhaps best illustrated by the reaction to non-siliceous materials, for silica is so irritating that it confuses the picture and fibrous asbestos is treated *in an absolutely unique manner.* Particles of coal, (Gardner[21] 1933), iron, gypsum, marble (Gardner and Dworski[22] 1922), carborundum (Gardner[23] 1923) etc, that succeed in reaching the terminal bronchioles tend to drift into the subpleural air spaces. There they encounter mononuclear phagocytes which migrate from the interior of the septa and move about on their surfaces Opinions differ as to the nature of these cells, many believe that they are epithelial in origin with special function because of their position, others, including the author, believe that they are




FIGURE 5. See caption on page 299

ordinary histocytes or connective tissue phagocytes that happen to have formed in the connective tissue separating air spaces There is also a theory that they are monocytes which have migrated out of the blood stream Whatever their origin they are freely moving monocytic cells which surround and engulf minute foreign bodies with which they come in contact They remain inside the air space for some time continuing to pick up any particle in their path When excessive concentrations of dust have been inhaled the phagocytes become over-distended and apparently paralyzed for they remain inside the alveoli for many months (Figure 5 *f*) When only an occasional dust particle must be removed the cells pick them up and wander about until they happen to reach the vicinity of a deposit of lymphoid tissue This may be at the point where an interlobular septum joins the pleura and the deep and superficial lymphatic systems communicate; it may be on the bronchial system at the distal end of

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FIGURE 5

Reactions to granite and chert dusts *a* Inhalation in guinea pig after 2 years exposure and 6 months in normal atmosphere Small focal collections of dust filled phagocytes scattered throughout parenchyma *b* Detail of one of the areas shown in figure 5a Collections of dust filled phagocytes in a group of air spaces Perilymphatic reaction in walls of small artery *c* Inhalation in guinea pig with chronic pneumonia Exposed for 13 months and then allowed to live for 22 months in a normal atmosphere *d* Liver of rabbit one year after intravenous injection of 3 3 gm granite particles (1 gm $SiO_2$) Collection of dust-filled phagocytes in portal connective tissue One of the very rare areas of cellular proliferation beside such a collection *e* Liver of rabbit two years after intravenous injection of the same dose of granite particles Necrosis has begun in the larger proliferative foci and there is proliferation in and about the small clumps of phagocytes *f* Liver of rabbit one year after injection of 1 gm of chert (50 per cent $SiO_2$) Heavy fibrosis in portal area with an atypical nonhyalinized nodule Two small focal areas of early necrosis in adjacent parenchyma

the alveolar ducts or at the bifurcation of arteries or veins. In any of these locations a small number of lymphoid cells surrounds a lymphatic vessel. The phagocyte leaves the air space and enters the lymphoid tissue. There it may either remain or pass through into the lumen of the lymphatic. In the latter event the current then carries it toward the hilum with the tracheobronchial nodes as its ultimate destination The cell may not reach this goal but may be held up in any of the lymph nodules along the way. Probably not all particles enter the lymphatics through the agency of phagocytes; a certain number may pass directly into these vessels. Small amounts of dust in practically normal lungs are most apt to reach the nodes at the root, as the quantity becomes larger there is a tendency *for accumulations to appear in the more* peripheral lymphoid tissues When excessive numbers of particles are inhaled over prolonged periods of time this orderly sequence of events does not follow. Many of the phagocytes do not succeed in entering the lymphatics through *the localized deposits of lymphoid tissue. They accumulate* in interstitial tissue in all parts of the lung The heaviest deposits occur in the areolar coats of blood vessels, in the interlobular septa and in the pleura, there are smaller amounts in the walls of the bronchi Since these are the locations where larger lymphatic trunks also occur the distribution is perilymphatic in type (Figures 4 *b* and 5 *f*) Whether dust-laden phagocytes pass into or out of the lymph vessels in these locations has never been demonstrated

The dust particles thus accumulated by phagocytic cells exert the same effect upon the tissue of the lungs that they do in other organs The injection experiments demonstrated that non-siliceous matter produces only very slight amounts of chronic inflammation The same is true in the lungs but since the number of particles collected in any focus is even smaller the reaction is apt to be less severe Gross inspection

shows linear and focal areas of pigmentation in the pleura, linear deposits along the trunks with focal collections in the associated lymphoid tissues and pigmentation of the tracheobronchial lymph node.

The amount of cellular reaction is insufficient to be recognized by palpation In microscopic sections there may be some proliferation of connective tissue but it involves so few cells that one is hardly justified in characterizing the reaction as *fibrosis* The changes produced in animals with pure non-siliceous substances are less severe than those encountered in human beings. The latter are generally due to much longer exposure and in most instances there is a certain amount of contaminating silica to accentuate the reaction.

*Free silica* Animals exposed to pure silica *in the form of* quartz or chalcedony develop lesions comparable to those *seen in human silicosis* (Gardner[24] 1932) *The inhaled particles are dealt with in the same manner as the non-*siliceous ones just described but the irritating silica produces changes which disturb the mechanisms for physiologic disposal The primary phagocytes are killed and degenerate, attracting polynuclear leucocytes An excessive number of new mononuclear phagocytes form in the alveolar walls and then migrate into the air spaces producing the condition which Irvine, Strachan and Simson[25] described as "bronchiolitis" (1930) Under the influence of silica these cells undergo epithelioid transformation and giant cells of the Langhan s type develop Stimulation also increases the rate of amoeboid activity so that the phagocytes move toward the lymphatic system with unusual rapidity The concentration of silica in and about the lymphoid nodules stimulates the local connective tissues The latter proliferate to form cellular nodules that replace the lymphoid elements. As the lesions increase in size they encroach upon the adjacent lymphatic vessels and ultimately compress their lumina Such

reaction takes place simultaneously in the intrapulmonary lymphoid tissues and in the tracheobronchial nodes. It is particularly well demonstrated in rabbits and albino rats, species which normally have well developed lymphoid tissues inside the lungs This sequence of events with the resultant impairment of the drainage system of the lungs constitutes the first stage in the development of silicosis.

After it is well established particles that are inhaled subsequently are perhaps eliminated less readily. At least it appears that they now tend to accumulate at various points in the walls of the air spaces. Here a repetition of the process produces a second generation of nodules which in this instance develop from irritation of the local connective tissues. With this stage true silicosis, which is characterized by "discrete nodules uniformly distributed throughout the parenchyma of the lung" may be said to begin. (Figures 1 *a* & 5 *c*) With concentrations of from 200,000,000 to 400,000,000 particles per cubic foot, mature lesions appear in animals in eleven or twelve months, with only 100 000 000, twenty-five to twenty-six months are required. Hard, pearly white nodules, 1 mm or less in diameter, project above the pleural surface and are scattered over its cut surface. The tracheobronchial nodes are greatly enlarged, very hard and exhibit a pearly lustre on section The hepatic lymph node is also involved although at a somewhat later period It is believed that this node does not receive its drainage directly from the lung but from the liver With heavy exposures many inhaled particles enter the blood stream which carries them to the liver and spleen In the latter they are retained and produce nodules, in the liver they remain only temporarily until the lymphatic system carries them out to the hepatic lymph node. Reaction in this node develops simultaneously with that in the spleen. Later, when the node is completely fibrosed the silica accumulates in the liver and visceral nod-

ules develop Other nodes in the animals are rarely affected. Hepatic lymph node involvement is common in human silicosis; abdominal visceral nodules occur but they are rare.

With concentrations of silica in excess of 500,000,000 particles per cubic foot the defense mechanisms of the lung seem to fail. Nodules develop simultaneously in the lymphatic system and in the walls of the air spaces In the latter there is also a diffuse, fine fibrosis and so many free phagocytes are killed that the picture has been described as a dust pneumonia Such reactions have been encountered in human beings exposed to excessive concentrations of almost pure silica. The writer observed them in a few sandblasters who had worked without adequate protection and in a number of men who had been employed in a notorious tunnelling operation in the United States (Gardner 1933)[26] A similar picture was found in men digging a tunnel for a sewer in Johannesburg (Irvine, 1935)[27]

It is of no moment to describe the minor variations of this reaction which appear in different species except to state that they exist and that their counterparts have been observed in different human subjects Such for example are the excessive tendency to necrosis followed by calcification which is the rule in rabbits, the more discrete and dense hyaline fibrosis of albino rats which is the usual form in human cases, and the less circumscribed foci of guinea pigs. The silicosis of fowls (Figure 5 *a*) is interesting because it probably develops without primary lesions in a well organized pulmonary lymphatic system It has been claimed that the lungs of birds lack such a system and although this statement has not been verified by detailed anatomic study, the inhalation experiments have failed to disclose evidence of it Even with moderate concentrations of dust, primary nodules seem to develop here and there in any portion of the

pulmonary stroma. Nothing comparable to tracheobronchial lymph node disease develops.

There is one feature which should be emphasized, namely, that the typical nodulation has been reproduced in all these various species without evidence of coexistant infection. Many writers still maintain that such complication is essential but these experiments constitute proof to the contrary. The silicotic lesions often simulate tubercles but repeated intracutaneous tuberculin tests on several large series of guinea pigs has definitely eliminated this possibility Other infections, notably chronic pneumonia and pulmonary abscess are frequent complications but the diffuse obliterating character of the reaction can be recognized and differentiated from pure silicosis

The cause of death in animals with silicosis is not always obvious Superimposed pulmonary infection is unquestionably the most common one Animals with the confluent, fine diffuse fibrosis from excessive exposures or those with extensive fibrosis from organizing pneumonia develop chronic passive congestion and die with dilated right hearts Rats with discrete nodulation have never shown cardio-vascular effects Renal lesions have never been observed in any species There has been nothing to suggest a general poisoning of all the tissues such as Collis and Yule (1933)[34] postulated Reactions develop only in organs whose anatomy is such that they retain appreciable collections of silica particles

## Inhalation of Mixtures of Free Silica with Other Substances

The inhalation of dusts composed of both free silica and other minerals has not always produced the same effects in the lungs of experimental animals that the same mixtures *appear to have caused in human beings* Not a few experimentors have reported the reproduction of lesions *resembling*

those of human silicosis but their illustrations belie their descriptions. In view of the unqualified success with pure silica these results are perplexing and demand an explanation. To dismiss them on the grounds of the inadequacy of the experimental method might well mean a lost opportunity to discover important principles which govern the development of pneumoconiosis.

It is conceivable that the experiments have failed because they have not actually reproduced the conditions of industrial exposure which have been defined by occasional sampling of the atmospheric dust Whether analysis of the rock being worked and counts of the number and size of the atmospheric dust particles are adequate to define the extent of the silica hazard may be questioned The composition of the dust in many industrial environments undoubtedly varies from hour to hour, at times it may contain much higher proportions of free silica than others In mining and in large plants like foundries the concentration of silica in the atmosphere depends upon the nature of particular operations. Many employees shift frequently from jobs involving exposures to high concentrations of almost pure silica to others where the silica is highly diluted with various substances Obviously the exact duplication of such conditions for experimental animals would be difficult and an explanation for the failures of experiments on this basis is highly theoretical.

Experimenters have not always appreciated the complexities of their problem Not infrequently someone reports that he has worked with a dust produced by grinding the rock that was being worked in the industry under investigation As already noted, this involves pitfalls, for to obtain a dust whose components are present in the same proportions in which they existed in the original rock it is necessary to reduce all of that rock to particles of the desired size If it is only partially ground the harder silica is resistant and may easily

be eliminated in subsequent separations. The dust itself should be analysed as a check. Even then there is sometimes a suspicion that the relationship of components is altered In the process of grinding it has even been suggested that the particles of silica become mechanically coated with the softer materials, but actual proof of such an effect is lacking.

Inhalation experiments, unlike those by injection, do not guarantee contact between the tissues and all of the component particles of a given dust. The rôle of the different factors which govern inhalation is of unusual importance in dealing with mixed dusts but they have hitherto received too little attention. Particles that are large and heavy, as is apt to be the case with quartz in improperly ground mixtures, settle more rapidly than smaller and lighter ones Different kinds of mineral particles suspended in the atmosphere do not necessarily remain separated, in fact they tend to agglutinate if they carry opposite electrostatic charges or possess hygroscopic properties The aggregates are heavy and remain suspended in the air for limited periods; they are also more readily caught by the protective mechanisms of the upper respiratory tract Selective exclusion of some component of a mixed dust could readily result from a greater degree of wetability and consequent adhesion to the mucous surfaces of the upper respiratory tract.

Some of the experimental 'failures' are probably the result of undue curtailment of the factor of *time* There is a tendency to think that by increasing the reciprocal factor of dosage the time may be shortened to conform with the limited span of life in ordinary experimental animals To what extent this is true awaits demonstration The injection tests, already discussed, indicate that certain substances like the iron oxide in chert, the silicates in granite and the calcium carbonate and calcium fluoride in crude fluorspar may inhibit the action of many times the effective dose of free silica. The duration of

such inhibition has not been established but the development of slight reactions in animals surviving for long periods suggests that it is overcome in time and that at least a modified silicotic fibrosis ultimately develops

Finally the experimenter may have defeated his purpose by insisting that results always be demonstrated in the lungs that are normal. This viewpoint is rational but one should not neglect to observe the effects of different dusts in the presence of infection for a great many human subjects have such complications. While it has been demonstrated beyond question that free silica can cause typical silicotic fibrosis in normal tissues, it has not been proved conclusively that apparently inert mixtures of silica and other substances may not produce progressive reaction in the presence of infection It will be shown presently that the inhalation of the natural mixtures granite and ferruginous chert has not produced nodular fibrosis in normal animals but that in ones with tuberculosis the reaction approaches the nodular type (Figure 4 *c*). These results may be due to a release of the inhibitory action of the non-siliceous elements in the respective dusts or they may merely reflect the greater activity from a high local concentration of silica caught in the inflammatory reaction to the infection

The purpose of this section is to demonstrate that the number, size and composition of the non-siliceous components of a mixed dust often determines the behavior of the particles of free silica Consequently it is necessary to describe in detail the conditions of the inhalation experiments to be cited as proof.

*Hematite contaminated with 6 per cent quartz* Guinea pigs, white rats and rabbits have been compelled to inhale excessive concentrations of this dust for periods of three and a half years They developed a pigmentation of the lungs and lymph nodes but none of them have shown anything even suggestive

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of a *silicotic reaction*. A group of surviving guinea pigs were removed to a normal atmosphere and allowed to live for another eight months. Then they were killed but autopsy revealed no progression of their pneumoconiosis

The average total concentration of dust at the breathing level inside the cages was 580,000,000 particles per cubic foot of air by light field count and by dark field, 6,250,000,000 Chemical analysis of the air-floated particles at the same point revealed 7 per cent of silica which by computation is equivalent to approximately 40,600,000 quartz particles (463,750,000 by dark field). Size frequency counts demonstrated that 95 percent of the total air-floating particles were less than 2 microns in diameter, none exceeded 3 microns Such concentrations were maintained for seven to eight hours a day, six days a week over the period specified The animals were not removed from the dusting room at night but remained in the same cages Counts taken during this period revealed between 3,000,000 and 7,000,000 particles per cubic foot by light field and 56,000,000 to 205,000,000 by dark field count. The total represents a very considerable exposure to silica over a period of three and a half years and yet the reaction in the lungs was that to a non-siliceous dust

It consisted grossly of linear and focal deposits of brick-red dust in the pleura, along the blood vessels and in the pulmonary and mediastinal lymphoid tissues. Microscopic examination showed compact collections of phagocytes filled with iron particles located inside of groups of air spaces or crowded into the areolar coats of arteries and the stroma of lymphoid tissues (Figure 5 *e*) Connective tissue proliferation was negligible except in the tracheobronchial lymph nodes where a limited amount of such reaction had developed *about very large collections of dust-filled phagocytes*

Chemical analysis of the lungs of animals killed at the end of the experiment, eight months after the cessation of the

dust exposure, demonstrated high ash values; the average for 3 animals was 22.88 per cent of the dried lung tissue. Iron constituted 76.15 per cent of this ash, the silica content was low, being only 4.89 per cent. After much shorter exposures to pure silica one finds 40 to 80 per cent of silica in the ash. Obviously so little silica had accumulated in the lungs of these guinea pigs exposed to hematite contaminated with 6 per cent of quartz that no silicotic reaction developed.

*Ferruginous chert containing about 50 per cent of silica.* Petrographically the dust employed consisted of a mixture of hematite, ferruginous chert, normal quartz and a small quantity of rutile. Chemical analysis revealed 36.8 per cent of free silica. The first experiment made with this dust failed to produce silicotic nodulation after exposures of eight hours a day over a period of one year and two months even though the total dust concentrations were excessive (light field counts over 1 billion and dark field counts in excess of 3 billion). Only a non-specific pneumoconiosis developed. Surviving animals allowed to live two and one half years in a normal atmosphere showed no progression of the reaction to dust. The histologic picture was essentially the same as that in animals exposed to hematite. Chemical analysis of their lungs at the end of the exposure yielded an average ash value of 10.25 per cent of which 5.8 per cent was silica. After two and a half years the silica content of the lung ash had decreased to 3.18 per cent. This was attributed to elimination of silica from the lungs by way of the lymphatics as the histological sections showed considerably more reaction in the lymph nodes at the end of the period. Unfortunately no chemical check was made by analysing the nodes.

The failure to reproduce the anticipated modified form siderosilicosis, after such long exposures to such excessive concentrations of dust demanded an explanation. It had been perhaps reasonably assumed that the animals were breathing

air containing at least 500,000,000 particles of silica per cubic foot by light field count and in other tests as low a concentration as 100,000,000 particles per cubic foot of pure quartz had repeatedly caused nodular fibrosis in about twenty-five months. A careful investigation of the conditions disclosed that while most of the particles in the dust received from the mine were less than 10 microns in diameter, only about 50 per cent of them were less than 3 microns Most of the silica particles were in the range between 3 and 10 microns. The behavior of this mixture in the dusting chamber proved surprising There was a very marked reduction in the percentage of silica particles between the hopper where the dust cloud was created and the air inside the animals' cages, never more than 6 feet away. The original material contained 46 8 per cent of silica; that which settled on the rafters showed 38.4 per cent while inside the cages the air-floating dust contained only 10 47 per cent.

The actual quantity of available silica of a size likely to produce much reaction was only 3 5 per cent greater than that in the hematite experiment cited above. In spite of the total concentration in excess of 1 billion particles per cubic foot there must have been only a little over 10,000,000 silica particles per cubic foot (40,000,000 by dark field count) in the animals' cages

In view of this experience another experiment was started with more of the same chert ground so that all of the particles were less than 3 microns in diameter. This proved expensive and time-consuming but 1500 pounds of such dust were finally obtained The total dust count by light field averaged 552,000,000 and by dark field, 1,983,000,000 particles per cubic foot of air. Inside the animals' cages 41 0 per cent of the particles were silica, giving an estimated concentration of available silica particles of 220,000,000 by light field and by dark field 793,000,000 per cubic foot of air. There had still

been a reduction of silica content between the hopper and the cages but with this very fine dust such reduction amounted to only 22 9 per cent instead of 36 3 per cent as in the previous experiment

As before, guinea pigs, rabbits and white rats were exposed to this dust and killed at intervals to observe the progress of their disease The experiment is not yet completed but after two years of treatment no sidero-silicosis has developed The lungs of the different species are heavily pigmented and the walls of air spaces containing masses of dust-filled phagocytes show slight chronic inflammatory reaction. (Figure 5 *f*) There is no fibrosis either in the lungs or the associated lymph nodes The lesions in the more sensitive white rats are essentially the same except for focal collections of phagocytes in the lymphoid tissues

Chemical analyses of the lungs of 4 guinea pigs exposed for two years showed only 15 5 per cent of ash of which 1 6 per cent was silica. Obviously the dust has not entered the lungs in any great quantity in spite of the excessive concentration in the atmosphere The silica content of the ash is surprising in view of the atmospheric concentration of 220,000,000 particles per cubic foot, 98 per cent of which were under 2 microns in diameter The conclusion seems inescapable that clumping or some other phenomenon either in the atmosphere or in the upper respiratory tract has retarded the inhalation of both iron and silica

To make certain that the silica in this mixture was capable of producing fibrosis it was tested by intravenous injection into rabbits Four of these animals were treated with 2 gm of chert, which would contain the standard dose of silica, four more received 1 gm. All members of both series developed a modified nodular fibrosis (Figure 4 *f*) although the reaction to the smaller amount did not progress as rapidly as that to pure silica. These findings were also confirmed by intra

peritoneal guinea pig tests Thus there seems to be no question but that the silica in the chert was in an active form.

To learn whether the iron would influence the action of silica that was already in the body another inhalation experiment was performed in which animals were exposed on alternate weeks, first to the hematite dust already mentioned and then to pure quartz The former was carried on in the same room and at the same time as the other hematite experiment where dust concentrations in excess of 500,000,000 particles per cubic foot of air were maintained The quartz concentrations were also high, averaging approximately 800,000,000 per cubic foot of air by light field count. The exposures were continued for twenty-two months and a group of survivors was kept for another six months in a normal atmosphere

After an exposure of one year the connective tissues about masses of dust-filled phagocytes in the lymphoid tissues and the pulmonary stroma had begun to proliferate. Such reaction continued to progress throughout the remainder of the exposure and during the period after removal from the dust house It occurred in focal areas scattered throughout all parts of the lungs The borders of these foci were not sharply defined , there was no central necrosis and no hyalinization of collagen which characterizes the mature reaction to pure silica in the guinea pig In the tracheobronchial lymph nodes, however, more typical silicotic nodules made their appearance after only six months of exposure , in all of the animals killed subsequently the lymph node lesions were larger and more numerous. The pulmonary foci resembled the early stages of the modified form of silicosis known as sidero-silicosis which one generally finds in hematite miners Had the exposures been prolonged further it is assumed that at least some of the pulmonary nodules would have developed characteristic central zones of laminated hyaline fibrosis.

These different experiments with mixtures of hematite and

varying quantities of free silica point very definitely to modifying action of the non siliceous component of the du The greatest effect seems to have taken place in the atmo phere where, because of clumping or perhaps other means much of the silica falls out of the atmosphere before it is wafted to the breathing zone of the animals a few feet away Perhaps more of the silica, caught in the clumps of iron particles is retained in the tortuous passages of the nose Comparatively little enters the lungs as demonstrated by the chemical analyses of these tissues Much longer exposures should ultimately produce reaction but of the modified type (siderosilicosis) observed when alternate exposures to pure silica and to hematite were employed

If iron dust is such a definite protector against silica it is natural to enquire why the miners who are exposed to the same form of dust should ever develop sidero silicosis Much of the miner's exposure might be compared to that of the guinea pigs which were compelled to breathe pure silica and almost pure hematite on alternate weeks The occupational histories which one obtains of so many years work in rock and so many in ore are only the roughest index of the actual character of the dust which a man breathes from day to day and hour to hour On the other hand, *time* may be the more significant factor Could these animals have been exposed for periods of ten or fifteen years enough silica would have undoubtedly entered their lungs to cause fibrosis The principles demonstrated by these experiments may explain the failure of approximately ¾ of most hard rock miners to develop silicosis even though they have apparently been exposed to the same amounts of dust as the other quarter of the men who do develop the disease These results also suggest the advisability of staggering employment in rock and ore to avoid prolonged exposure to dust of high silica content

*Artificial mixtures of calcined gypsum and pure silica* At

tention was called to a similar protective action by gypsum dust in a clinical study of a group of workmen. A few of these men had been engaged in grinding calcined gypsum with buhrstones and two of them had been chipping the stones in the same room for periods of five and ten years respectively This is a notoriously hazardous operation. Dust counts taken as they worked were high and the concentration of silica was such that one predicted a hazard. Examination of the men, however, showed no evidence of silicosis.

Another group of workmen in the same industry make 'sanded plaster' by mixing sand with calcined gypsum. In their department the dust counts were very high and chemical analysis revealed too much silica in the atmosphere. The size of the silica particles was unfortunately not determined While they were small enough to have remained suspended in the air they had been produced from a washed sand and should theoretically have been fairly large, probably greater than 3 microns in diameter. Six men were found working in this department who had been employed from five to twelve years. One of them had worked exclusively on a machine packing sanded plaster for a period of ten years. A roentgenogram of his chest showed no abnormalities whatsoever; the films of the other five were either negative or revealed moderate degrees of exaggeration of the linear markings. In spite of the absence of evidence of nodular silicosis the conditions of employment were deemed dangerous and it was assumed that the period of employment had not been long enough to produce changes demonstrable by roentgenogram To test this supposition the gypsum company was asked to have examinations on all men engaged in making sanded plaster in its various plants throughout the state The company cooperated and brought together 17 men who had been employed at this work for periods varying from ten to thirty-three years. All of them were given complete physical examinations, including roent-

genograms but none showed even a suggestion of silicosis One had a normal film and the others exhibited the usual degrees of linear exaggeration to be expected in persons of their age The intensity of this reaction did not increase with the duration of exposure so that it might or might not have been produced by the dust that the men had inhaled

Here was clinical evidence of protective action against silica provoked by another non siliceous material Examination of the dust in the mills demonstrated that it was composed of good sized clumps which settled out of the atmosphere rapidly It was not possible to determine the relative proportions of silica and gypsum in any one clump nor the spacial relation ships between the two kinds of particles But it was proven that clumps existed and as it is known that silica particles carry a negative charge while gypsum has a positive one, it was assumed that the electrostatic effect was responsible for the clumping.

From the clinical standpoint it seemed that the protection from gypsum was presumably much more complete than that conferred by iron for no case of even a modified form of silicosis was discovered in the gypsum industry To test the protective action of this substance further another series of animal experiments was made

A group of guinea pigs, injected intraperitoneally with a buffered suspension of finely ground calcined gypsum particles developed no fibrosis although there was some necrosis in the centres of the largest masses of dust In animals killed during the ensuing year these necrotic foci had become calcified This experiment indicated that no fibrosis and practically no other reaction could be expected from inhaled gypsum.

Nevertheless 100 guinea pigs were exposed for two years in a dusting room to concentrations of gypsum dust averaging 118,000,000 particles per cubic foot by light field and ap-

tention was called to a similar protective action by gypsum dust in a clinical study of a group of workmen. A few of these men had been engaged in grinding calcined gypsum with buhrstones and two of them had been chipping the stones in the same room for periods of five and ten years respectively This is a notoriously hazardous operation. Dust counts taken as they worked were high and the concentration of silica was such that one predicted a hazard. Examination of the men, however, showed no evidence of silicosis

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Nevertheless 100 guinea pigs were exposed for two years in a dusting room to concentrations of gypsum dust averaging 448,000,000 particles per cubic foot by light field and ap-

proximately twice this amount by dark field count. The particles were fine, 96.3 per cent being less than 3 microns in diameter. No significant reaction developed. Most of the dust apparently dissolved for only occasional phagocytes were found scattered through the air spaces of the lungs. In the tracheobronchial lymph nodes a moderate amount of chronic inflammatory change developed about clumps of phagocytes containing less soluble particles. The latter reaction progressed somewhat in successive animals but it never attained a degree worthy of the name fibrosis.

Having demonstrated that calcined gypsum itself causes no effects of importance, the action of gypsum in combination with quartz was then investigated. In the first experiment, equal volumes of pure quartz * and pure gypsum dusts were placed in the agitating hopper in one of the experimental dusting rooms. The rotating paddle in this apparatus soon mixed the two dusts very thoroughly. A chemical analysis showed 49.7 per cent of $SiO_2$ and 45.8 per cent $CaSO_4$. The average concentration of the dust generated with this mixture was 460,000,000 particles per cubic foot of air by light field count and 900,000,000 by dark field. A size frequency count revealed that 97.8 per cent of the air-floated particles were less than 3 microns in diameter. Theoretically this dust cloud should therefore have contained 223,500,000 particles of silica less than 3 microns in diameter.

Analyses revealed that, as in the case of mixtures of iron and silica there had been a very marked reduction of the amount of silica in the air which reached the interior of the animals' cages. Chemical analysis of dust collected with an

* The quartz employed for the purpose consisted of 100 per cent normal quartz as determined by chemical and petrographic analysis. Most of the particles were less than 10 microns in diameter. It was the same material used in the experiments previously discussed and its capacity to cause fibrosis at the time it was used was checked by two other experiments that were being carried on simultaneously.

impinger at this point showed an average silica content of only 29.6 per cent, a reduction of over 21 per cent from that of the material in the hopper. Dust settled on top of a cage 6 feet above the floor showed a higher percentage of silica than that in the hopper, the figures being 53.7 and 49.7 per cent respectively. As a further check, counts of the number of particles in the original alcoholic suspensions of air borne cage dust were compared with those on another portion of the same suspension which had been treated with acid to dissolve the gypsum. This method revealed only 42,750,000 acid insoluble particles per cubic foot by light field and 127,750,000 by dark field count. Thus there can be no question but that much silica had been removed from the atmosphere by the time it reached the interior of the cages. By actual count the number of particles was over five times less than the estimated value. The discrepancy between the computed and the actual number of particles is probably due to the small weight of very fine silica particles.

To demonstrate the extent to which aggregates had formed in the atmosphere a dry slide was placed inside of an empty cage for a period of 15 minutes and a size frequency count of the clumps settling on it was made. Their number in various sizes was as follows:

| *Size* | *Number* |
|---|---|
| 0 – 5 microns | 52 clumps |
| 5 –10 " | 18 " |
| 10–20 " | 17 " |
| 20–30 " | 7 " |
| 30–40 " | 5 " |
| 40–50 " | 1 " |

The maximum size is not very large but when similar preparations were made outside of the cages, where the dust

settled directly on the slides, the clumps were so large and numerous that they could neither be counted nor measured But all of the figures reported are materially greater than the maximum of 3 microns for the individual particles.

Guinea pigs kept in the atmosphere just described for periods of twenty-five months and survivors removed to normal atmospheres for a subsequent period of six months failed to develop typical silicotic nodulation of the lungs. Until the exposures had been continued for a period of seventeen months their lungs exhibited no localized reaction whatsoever, the response consisted only of phagocytes scattered widely through the air spaces. But then the cells began to collect in masses within the terminal bronchioles and to migrate into the lymphoid tissues in increasing numbers. Proliferative reaction in the pulmonary framework ensued but not in the form of nodules; there was merely a thickening of the alveolar septa adjacent to clumps of intra-alveolar phagocytes (Figure 5 *d*) Similar pulmonary changes were found during the next eight months. In the tracheobronchial lymph nodes hyaline silicotic nodules were discovered after twenty-three months of exposure and continued to occur in most but not all of the animals killed subsequently. In members of the series killed after removal from the dusting room the pulmonary reaction increased in intensity; a modified cellular type of nodule developed but usually it did not undergo necrosis nor exhibit characteristic hyalinization. Its borders were always ill-defined. In only 2 guinea pigs killed during the first month after cessation of the exposure were any hyaline foci discovered and these were few in number; in the cases examined later such nodules were absent.

Through an unfortunate oversight no chemical analyses were made of the lungs of these animals but the anatomic evidence points to a marked reduction in the amount of silica inhaled It took twenty-three months for enough to be con-

centrated in the tracheobronchial lymph nodes to cause mature reaction, four times as long as is required with pure silica. Within the lungs enough never accumulated to produce nodulation except in two animals which seemed to respond more readily than their fellows. The lesions in the exceptional cases were sufficiently well marked to raise the question as to whether longer exposures would not ultimately produce nodular fibrosis in a majority of the animals.

To answer this question another inhalation experiment was started but it has not yet been completed. Less silica was employed in the second mixture in the hope that the observations might be extended over a longer period of time. 1 volume of quartz and 3 volumes of gypsum were placed in the hopper of the dusting device. Chemical analysis after mixing revealed 31.4 per cent of $SiO_2$ and 62.4 per cent of $CaSO_4$. The silica content of this dust showed a similar reduction from the hopper to the breathing zone. In the latter position an analysis of an impinger sample revealed only 17.1 per cent of silica, a decrease of 14.3 per cent. The average total concentration of dust in the atmosphere inside the animals' cages was 553,750,000 particles per cubic foot of air by light field count; under dark field the figure was 1,577,000,000. By computation the number of quartz particles at the breathing zone should be 94,000,000 per cubic foot or on the basis of the dark field count, 367,667,000.

Guinea pigs and white rats have now been exposed to this mixture of silica and gypsum for twenty months and the results are essentially the same as those of the previous experiment. Silicotic reaction has not appeared in the lungs. Chemical analysis has demonstrated less than 5 per cent of ash and correspondingly low figures for silica (approximately ... per cent of the ash). For relatively pure dusts like silica and hematite the amount of ash is three to five times as gr... These findings indicate that not much dust of any typ...

entering the lungs and that the silica in particular is less than would be anticipated in view of the atmospheric concentrations described

To ascertain whether gypsum dust already present in the lungs will alter the reaction to silica inhaled independently, one group of guinea pigs were exposed on alternate weeks to pure gypsum and pure silica A second group that had inhaled gypsum for two years was placed in an atmosphere of pure quartz on a schedule similar to those previously described. In neither experiment did the gypsum have any apparent effect upon the development of silicosis.

The experiments with gypsum dust seem to demonstrate the principle of protection, which is probably responsible for the lack of evidences of silicosis in the men exposed in localized parts of the plants. Although the industrial atmospheres contained quantities of silica which were considered hazardous, not enough of this substance appears to have entered their lungs to cause reaction demonstrable in a roentgenogram. Had the buhrstone grinders not been inhaling gypsum along with silica dust it is hardly likely that they could have escaped.

*Granite.* A final example of the same principle is probably offered by an experience with granite dust from Barre, Vt The dust employed contained 35 per cent of free silica as quartz Inhalation experiments [28] (1920) were made many years ago, before the necessity of evaluating all of the factors had become apparent. The dust for the test, obtained from the collector of an exhaust system in a cutting shed, was composed of a mixture of fine and coarse particles. Average dust counts by light field illumination at the breathing level of the animals revealed 957,000,000 particles less than 10 microns in diameter, and of these 165,000,000 were less than 1.5 micron in diameter At the time 50 guinea pigs were exposed in boxes 6 x 3 x 2 feet in diameter for eight hours a

day from which they were removed at night The longest period of exposure was two years, a few animals were allowed to live as long as six months after cessation of the dust exposure.

The maximum reaction to this amount of granite dust was definitely greater than that to hematite but still it did not even simulate the nodular response to pure silica (Figures 4 *a* and 4 *b*) It consisted of dust-filled phagocytes collected in focal areas within groups of air spaces or in the areolar tissues along the lymphatic trunks The connective tissues had been somewhat stimulated for the external coats of the arteries and the larger septa were thickened though by a cellular, rather than a fibrous type of reaction In the lymphoid tissues the granite exerted more effect In a few of the longest surviving animals the tracheobronchial nodes contained isolated nodules whose centres were beginning to degenerate In the lungs nodules never developed except in the presence of infection. Even then the focal lesions were not typical of silicosis They consisted of masses of cellular connective tissue from whose margins strands of a similar nature radiated into the surrounding stroma (Figure 4 *c*) There was no central area of necrosis, no hyalinization of collagen and no tendency of concentric lamination Such foci were not tubercles for they developed in non-tuberculous as well as tuberculous lungs, but neither were they typical silicotic nodules It was assumed that characteristic central hyaline zones might ultimately appear but this did not occur within the two and a half year period of observation.

These experiments were completed long before facilities for accurately defining the number and size of the free and combined silica particles in the atmosphere were available but the records indicate that the dust employed was not deficient in fine quartz Chemical analysis of the lungs were not made but re examination of sections in polarized light

today discloses myriads of doubly refractile particles that look like quartz The more recent injection experiments with the same kind of granite indicates an explanation for another apparent failure; the action of the quartz was retarded by the other components of the dust. In normal animals the phagocytes were not stimulated so that they failed to concentrate the quartz in focal areas; in tuberculous animals the infection caused an appreciable tendency to localization. But even when this had occurred, the silicates prevented the free silica from exerting its usual action upon connective tissue cells There was some stimulation but it would undoubtedly have taken many more years of contact before typical silicotic foci developed.

These observations are in accord with clinical experience among the granite cutters at Barre In non-infected individuals nodular fibrosis only develops after exposures of twelve to twenty years, even though the dust concentrations have been excessive. In roentgenograms of these men Russell, Britten, Thompson and Bloomfield[29] (1929) reported that linear reactions were much more common than nodular ones. Such shadows would be cast by the sheaths of perilymphatic cellular connective tissues about the blood vessels Only this type of change was present in several post-mortem specimens which these investigators submitted to the writer for pathological study The modifying action of infection was well demonstrated in another pair of lungs subsequently received from the same community. There had been a self-limited tuberculous infection which was localized to a portion of one lower lobe. About this lesion were a number of small typical silicotic nodules. In the other portions of the lungs the reaction was not nodular but linear and perilymphatic in distribution

Granite dust would therefore seem to be a material containing inhibitors which are capable of preventing quartz

from acting on the tissues for a considerable period of years. The effect of these substances is apparently exerted within the tissues in a manner not yet understood Infection tends to neutralize the inhibition so that the quartz can act more rapidly. Ultimately the protective action is overcome and a more or less modified silicosis develops It would be profitable to determine quantitatively the amount of feldspar or mica or other unnamed substance which is necessary to neutralize the action of a unit quantity of quartz.

From the experimental and clinical observations that have been cited it no longer seems reasonable to assume that the silica hazard is only proportional to the amount of free silica in an atmospheric dust The other substances present in combination with the silica modify its action Some of them, like iron and gypsum, prevent its inhalation ; others, like the [illegible] of granite seem to prevent it from affecting the [illegible] of metallic aluminum has not [illegible] suggest [illegible] be [illegible] ern [illegible] that it may fall in the [illegible] quantitative relationship [illegible] done to evaluate the quantitative relationship of the action of inhibitor substances on silica but that they exist and exert a very definite influence is the burden of this argument.

## ASBESTOS

The only form of asbestos tested by inhalation in animals has been chrysotile but there is every reason to believe that the other fibrous silicates would produce similar effects for in human beings the reactions to all members of this group are apparently indistinguishable When guinea pigs, albino rats and rabbits were exposed simultaneously to chrysotile dust of an average concentration of 43,000,000 particles per cubic foot of air only the guinea pigs developed significant reactions. ( Gardner and Cummings 1931 ) [30] Apparently the

upper respiratory protective mechanisms of the rabbits and rats were adequate to exclude fibrous foreign bodies. After about seventy days exposure, asbestosis bodies had developed about fibres of chrysotile within the air spaces of the guinea pigs lungs. These asbestosis bodies closely simulated those found in human beings but were considerably smaller. They presented the same golden-yellow color, the club-shaped terminal swellings and the transverse segmentation of the iron-containing deposit upon the mineral fibres. They were located very largely in the alveoli opening along the sides of the respiratory bronchioles. Many of them were free; some were surrounded by giant cells of the foreign body type As the inhalation continued more of them formed in the same location where they either remained or penetrated the wall of the bronchiole. None could be found in the intra-pulmonary or tracheobronchial lymphoid tissues. Only the non-fibrous particulate elements of the dust were transported to the lymphatic system

With further exposure the walls of these air spaces, penetrated by the fibrous foreign bodies, gradually became thicker due to an infiltration with mononuclear leucocytes. After fifteen months a moderate amount of fibrous tissue had formed, which surrounded the bronchiole like a sleeve At the time it was believed that contraction of this fibrous sleeve would close the tube and produce an atelectasis of the peripheral air spaces Study of the further stages of the disease has failed to confirm this prophecy Instead, the reaction seems to extend peripherally along the bronchiole apparently because the foreign bodies are deposited further along its course This, in turn, seems to be due to the smoothing of the inner surface and perhaps the loss of elasticity of the walls of the tube. As more fibres are inhaled they apparently pass through the smooth, inelastic bronchiole and are deposited in the alveolar ducts given off at its terminal end. These tubes in

their turn respond to the local irritation and their walls become fibrous In time the still more distal atria are similarly involved Although appreciable reaction in the alveolar sacs and alveoli has not been observed within two years and nine months of exposure, it now seems more probable that these structures are eventually involved by the process of peripheral extension

In another group of guinea pigs it was demonstrated that the reaction to inhaled asbestos dust is not a progressive one The animals were exposed to the same concentrations of chrysotile as used in the first test for a period of eight months and twenty days They were then removed to a normal atmosphere and members of the group were killed at intervals during a subsequent period of three years At the end of the exposure, chronic inflammatory reaction had developed in the walls of the respiratory bronchioles During the next three months this process gradually progressed to form a slight amount of fibrous tissue Thereafter the reaction was, if anything, retrogressive in character At the end of the three year period there were very thin sleeves of fibrosis about the bronchioles but no extension into more peripheral air spaces had occurred

In 1937 a third experiment with chrysotile was begun This time the asbestos fibres were ground in a steel ball mill until practically none more than 3 microns in length were left It was anticipated that, if the irritation were of a chemical nature, the reaction should proceed more rapidly Average con centrations of 175,000,000 particles per cubic foot ( light field count ) are being maintained in the dusting room Thus far *the response to 4 times the concentration of a much finer dust has been less pronounced than that obtained in the first ex periment* Instead of being held on the irregular surfaces of the respiratory bronchioles, the particles have passed immediately into the terminal air spaces The very short fragments

of asbestos are surrounded by a typical golden-yellow coating and have the appearance of minute asbestosis bodies. Practically all of them are found within the bodies of mononuclear phagocytes. Only a few can be detected in the interstitial tissue. Most of these seem to be intracellular. Some have been transported to lymphoid tissues. Evidence of chronic inflammatory reaction in the terminal bronchioles and other parts of the pulmonary stroma are practically absent Compared with animals exposed for similar periods to the longer fibres of chrysotile in the first experiment, the picture is very different and more nearly resembles that produced by inert dusts

These experimental observations seem to be indicating that asbestos fibres cause fibrosis in the lungs because they act as mechanical irritants in an organ whose function involves considerable movement. The long fibres cannot effectively be removed by phagocytes and the lymphatic system, they penetrate the walls of the bronchioles and with each respiratory effort they continue to injure adjacent cells. The development of a protective coating about them must materially reduce the friction but as long as the inhalation continues new uncoated fibres are constantly accumulating to keep up the irritation. This is inferred from the lack of progression after the exposure has ceased If the irritation were chemical in nature one would expect that it would develop more rapidly with finer than with large fibres, that it should be produced by injection into organs other than the lungs, as is the case with free silica, and that serpentine, whose composition is identical with that of chrysotile, should also cause fibrosis. But none of these effects have been observed Final proof of the mechanical action of asbestos upon the lung is still lacking; it remains to demonstrate that the inhalation of pure, very long fibres of chrysotile will produce results even more quickly than the dust used in the first experiment, which contained only a limited amount of such

material. However, the findings in the various experiments already finished all point to mechanical irritation. Since summarizing these observations a paper by Sundius and Bygdén [31] (1937) has come to hand. As a result of elaborate chemical and petrographic analyses of asbestos dust and asbestosis bodies these writers have reached the same conclusion.

The experimental exposures have thus far failed to reproduce the widespread fibrous lesions which have been observed in a few of the human cases of asbestosis Where the latter are uniformly distributed throughout the lungs but retain their characteristic fine, interstitial arrangement they may be considered as the result of prolonged and intensive exposure But where a human case reveals localized areas of massive fibrosis, it seems most probable that infection has complicated the picture. The majority of tuberculous infections in guinea pigs with asbestosis tend to spread for a time but subsequently heal with the formation of considerable areas of fibrosis Likewise, accidental non-tuberculous infections of the lungs may heal and organize to produce fibrosis The similarity between these foci of healed tuberculous and non-tuberculous infections in experimental animals and certain massive areas of fibrosis in the human cases strongly suggests a common origin. Furthermore, in guinea pigs the interjection of the factor of infection causes the fibrosis to become progressive and it is felt that the same may be true of human cases This may explain the belief, entertained by certain authorities, that asbestosis is itself a progressive disease.

## Infection and Pneumoconiosis

One of the important contributions to the study of pneumonoconiosis in recent years is the demonstration that silicosis specifically predisposes to tuberculosis and that the combined action of silica dust and the tubercle bacilli produces a chronic progressive disease which is responsible for

most of the cases with marked disability. The group of investigators in the Miners' Phthisis Medical Bureau at Johannesburg[32] (1930) clearly differentiated the respective rôles of the factors of infection and dust Collis[33] (1915) in his Milroy Lecture of 1915 showed that pneumoconioses other than silicosis were not associated with an excess incidence of tuberculosis although they might predispose to pneumonia Recent work in this country and in Great Britain[34] is now indicating that the latter is probably true only of the bronchopneumonias; the incidence of lobar pneumonia seems to be little higher in persons with pneumoconioses than in others.

In considering this phase of the subject, a definite distinction should be made between slight or healed infections which play only an etiologic rôle and clinically active infections which modify the course and symptomatology of silicosis The former may merely damage a localized area in the lungs so that abnormal quantities of dust are retained. Even though the infection is completely healed, the formation of scar tissue or the less perceptible alterations of the local lymphatic circulation modify the deposition of dust and the resultant form of the pneumoconiosis. On the other hand, the inhaled dust may alter native susceptibility to bacteria and the course of resultant infections in the lungs

Non-tuberculous pneumonias develop spontaneously in animals inhaling dust with annoying frequency but so little is known about the mechanism responsible for these epizootic conditions that it is hard to evaluate the rôle of the dust It seems highly probable that other environmental factors in the dusting rooms play considerable part in the spread of infections At least their frequency is not very much greater where irritating free silica is involved than in the case of inert materials, like gypsum or marble. The pneumonias are probably more chronic and more frequently organized when

they complicate silicosis than other pneumoconioses but this result is by no means invariable On the other hand such infections may modify the response to the inhaled dust. A purulent bronchitis opposes such a barrier to inhaled dust particles that very few of them enter the terminal air spaces Preexisting foci of chronic pneumonia entrap unusual quantities of dust and the resultant concentration of particles sometimes causes much more reaction than occurs in normal portions of the lung Silica accumulating in an inflammation starting to organize accelerates the process and produces diffuse obliterating fibrosis Acute pneumonias superimposed upon well established pneumonocomosis may be unusually fatal

For the study of tuberculosis several controllable technical procedures are available. The writer has found that in silicotic animals primary inhalation infection with a particular attenuated strain of tubercle bacillus gives results comparable to those observed in human beings He has therefore adopted this method as a standard procedure for testing the influence of other dusts on tuberculosis His work has been criticized because it attempts to compare the results of a *primary* infection in animals with the chronic disease arising from *reinfection* in the human subject The validity of this objection is fully appreciated and the original program of investigation contemplated artificial reinfections in animals previously sensitized However, repeated attempts demonstrated that the vaccinating infection would generally become progressive in any animal exposed to silica regardless of the site of administration Since endogenous reinfection occurred so regularly there was little point in making artificial reinfections While the same difficulty is not encountered in animals exposed to non siliceous dusts it seemed advisable to adopt a uniform technique for testing so that the results might be compared

The particular strain employed is known everywhere as $R_1$. It possesses a limited but rather constant virulence for guinea pigs which has been maintained over a period of forty years or more. Its action after either injection or inhalation is predictable and hence it is a better testing agent than many others which have been tried. When guinea pigs are made to inhale a vapor containing small numbers of $R_1$ tubercle bacilli, they develop a multiple primary complex comparable to the so-called "childhood infection" in human beings. In normal animals the lesions consist of a variable number of isolated discrete tubercles, all less than 2 mm in diameter, located in the subpleural alveoli together with foci of tubercle in the tracheobronchial lymph nodes. Such tubercles almost never spread but after progressing to the stage of caseation they begin to heal Within two years time even microscopic traces of the lesions may have completely disappeared (1922).[35] The only evidence of the infection may be a persistent hypersensitiveness to tuberculin (Figures 6 *a* to 6 *f*)

The course of an $R_1$ inhalation in a guinea pig exposed to dust may be quite different from that just described. The nature of the modified response depends upon the type of the pneumoconiosis concerned and the stage of its development when infection supervenes. Animals infected after several months exposure to dust of any type develop more tubercles and many of them are located in the deeper as well as the subpleural portions of the lungs; the course of the resultant infection is determined by the character of the dust. Under the influence of inert, soluble dusts like gypsum or marble the tubercles rarely spread and most of them heal by the normal process of resolution; a few may persist as localized scars, sometimes surrounding a focus of calcification (Figure 7 *a*) The silicate, chrysotile asbestos and the hematite mentioned above often cause a temporary local *extension*

of the infection, but in most animals it subsequently heals with the formation of more extensive areas of fibrosis. (Figures 7 e and 7 f) The natural mixtures of free silica and iron oxide, chert, and the artificial mixtures of gypsum and quartz produce even more marked spreads of the infection. Such reaction usually heals with extensive scar formation (Figure 7 d) but in some of the animals chronic, slowly progressive cavities develop which persist for several years. Granite with its 35 per cent quartz content may also cause chronic progressive tuberculosis but only after prolonged periods of exposure to the dust. Pure silica, and for unexplained reasons, the carbide of silicon (SiC) almost invariably produce a progressive type of infection.

If the tubercle bacilli are inhaled at the beginning of an exposure to pure silica dust the lesions pursue a normal course for a period of about five months. Then they begin to spread, at first by local extension into the adjacent lung tissue, then by ulceration into a bronchus with resultant dissemination through the air passages to all parts of the lung and finally by way of the blood stream to the abdominal viscera. Cavities form at the site of many of the original subpleural foci and often in secondary lesions throughout the lungs. The generalized reaction is at first exudative in character but soon becomes proliferative with the formation of tuberculous granulation tissue. Organization of the latter results in a widespread fibrosis which completely obliterates all of the normal pulmonary architecture. The animals appear well and suffer no loss of weight or appetite for many months but they become increasingly short of breath and finally die emaciated with all the signs of intoxication. Those with widespread pulmonary fibrosis often present the picture of right-sided heart failure at autopsy.

If the infection is administered at the end of a silica exposure and the animals are then removed to a normal at-




FIGURE 6 See caption on page 333

mosphere the resultant tuberculosis becomes increasingly acute as more and more silicotic fibrosis develops Infections after the first four or five months of dusting are generally quite chronic and their course resembles that just described Administered after six to ten months of dust exposure, the tuberculous infection runs a more rapid course with frequent fatalities; after twelve to fourteen months in silica dust, $R_1$ inhalation produces an acute pneumonic tuberculosis which in most cases terminates fatally within a period of three months. (Figure 7 *b*) Occasional resistant members of the last group may survive to develop more chronic lesions with large cavities and tuberculosis throughout their bodies

If, on the other hand, the inhalation infection is given first and healing is allowed to begin before exposure to silica dust the tubercles are susceptible to reactivation as long as they contain viable organisms (1929).[36] For this to occur it is essential that appreciable quantities of dust be localized in the immediate vicinity of the tuberculous focus Whenever this occurs mobile phagocytes transport silica particles into the caseous centres of the lesions, the bacilli are stimu

FIGURE 6

The course of $R_1$ inhalation tubercle in normal guinea pigs *a* Low power photograph to show three small isolated tubercles beneath the pleura on the right side The tracheobronchial node on the right is slightly enlarged due to simultaneous involvement *b*. An $R_1$ tubercle at its maximum stage with a central zone of caseation *c* Beginning healing The tubercle bacilli are largely dead, the caseous zone is shrinking and the proliferative zone has become fibrous and is contracted *d* The caseous zone is reduced to a small area at the upper right Contraction of the surrounding fibrous tissue is causing emphysema Note the associated dimpling of the pleura *e* All trace of caseation has disappeared Thickened septa separate considerably dilated air spaces The pleura is still thick *f* Practically all that remains after two years is the localized pleural thickening and some distortion of the underlying air spaces




FIGURE 7 See caption on page 335

lated to multiply and an extension of the infection ensues. The result is a chronic spreading tuberculosis whose anatomic manifestations are essentially the same as those in the first example of infection simultaneous with the outset of exposure of silica (Figure 7c).

It would appear that the bacilli multiply in areas where a sufficient quantity of silica particles has accumulated. But the mere presence of the silica is not the only essential; the mineral must have had time to react with the tissues and produce an appreciable amount of necrosis. The importance of the latter is well demonstrated by a technique devised by Kettle[37] (1924). In white mice, he showed that blood-borne tubercle bacilli, which happened to localize in an area of necrosis that had been produced by subcutaneous injection of finely divided silica, multiplied much more rapidly

FIGURE 7

$R_1$ inhalation in guinea pigs exposed to various dusts. *a.* Healing of an $R_1$ subpleural tubercle by calcification in a guinea pig exposed to pure gypsum dust for 5 months, then infected and allowed to remain in the dusting room for 21 months before it was killed. *b.* Acute, tuberculous pneumonia in a silicotic guinea pig infected with $R_1$ tubercle bacilli. Exposure to pure chalcedony one year, removed from dust room, infected by inhalation, death from tuberculosis in 41 days. *c.* Chronic silico-tuberculosis with cavity formation resulting from reactivation of a partially healed $R_1$ inhalation infection. The tuberculosis had been present 10 months before the exposure to pure quartz was begun; the guinea pig died after 7 months in the dust house. *d.* Chronic sidero-silico-tuberculosis in a guinea pig. Animal exposed to ferruginous chert for 3 months, infected by inhalation of $R_1$ tubercle bacilli and then returned to dust house for 12 months. *e.* Healing by fibrosis after local extension of $R_1$ tubercle in guinea pig infected by inhalation and exposed to asbestos dust for 5 months. Note that most of the tubercles are situated in the deeper portions of the lungs rather than in the subpleural regions as in normal control animals. *f.* Higher magnification of one of the fibrous tubercles shown in figure 7e. This lesion is much larger and more fibrous than those produced by either the infection or the dust alone.

than in other portions of the body. No proliferation occurred unless a period of four or five days elapsed between the 2 injections He also demonstrated the specificity of the silica reaction by producing subcutaneous foci of necrosis with other irritants, like turpentine, and showing that in them there was no unusual multiplication of the bacteria Vorwald and Landau [20] (1937) used the same method and demonstrated that the injection of non-siliceous dusts would not cause tubercle bacilli to multiply. With various silicate dusts their results were so inconstant that they could draw no conclusions as to the effect of any of these minerals

Other experimental methods are applicable to demonstrate the stimulating action of silica upon tuberculous infection Silicotic rabbits injected intravenously with human type of tubercle bacilli, which are ordinarily of low virulence for this species, develop progressive tuberculosis The infection localizes in and about the preexisting silicotic nodules and produces a collateral zone of tuberculous reaction. Caseation invades and often destroys the dense silicotic fibrosis White rats, which are naturally immune to tuberculosis and rarely develop localized tubercles, become susceptible after they have inhaled enough silica dust. Tubercles form in their lungs, both in the immediate vicinity of the nodules and in the intervening tissues Intratesticular subcutaneous and intraperitoneal injections of both tubercle bacilli and various dusts have been tried but the resultant lesions are so complicated that analysis is difficult

It now seems apparent that the reaction of silica upon the tissues furnishes a medium upon which tubercle bacilli grow with unusual rapidity But such growth is not necessarily continuous ; in newly-formed lesions the organisms are very numerous and readily demonstrated in stained sections or smears, but in old foci bacilli are often rare or entirely absent Subinoculation into guinea pigs usually demonstrates

their presence in experimental lesions. The writer has studied four advanced human cases with typical cavities and every histologic indication of tuberculosis in which even guinea pig inoculation of caseous cavity contents failed to reveal evidence of living bacilli.

Several investigators have attempted to demonstrate that the addition of silica in various forms to artificial culture media enhances the growth of the tubercle bacillus. Price[38] (1932) reported favorable results but Kettle[3] (1930) and the writer have both failed to note any result other than an early acceleration of growth. More recent experiments by Vorwald, Dworski and Steenken in the Saranac Laboratory have indicated that under certain conditions the amount of growth in the presence of silica is excessive. However, it has not been possible to obtain the same effect in every series of tests and these investigators are still attempting to discover the conditions responsible for their favorable results.

The exact cause of the stimulation awaits demonstration. Some, like Cummins[39] (1934) have ascribed it to obstruction of lymphatics which interferes with free elimination of the bacilli from the lungs. Others have believed that the silica itself directly stimulated the bacteria. The writer is of the opinion that the chemical products liberated by action of silica on the tissues provides a medium particularly favorable for growth. This view is based upon the presence of such large numbers of bacilli in fresh, necrotic silicotic foci and their paucity in lesions which are old and without degenerative changes.

The application of these experimental results to the interpretation of tuberculous manifestations in human silicotic subjects may be somewhat as follows. Activation of a preexisting primary complex of first infection such as has been described in animals does not often occur in human beings because such lesions are generally healed and sterile in per-

sons who have reached the age of industrial employment. However, very occasional examples of such reactivations have been followed in young persons, presumably ones infected for the first time in late adolescence. Such cases prove the possibility of affecting the primary complex but their infrequency makes it at least debatable as to whether the presence of the calcified primary complex constitutes reasonable grounds for rejection from employment. It seems most unlikely that the small calcified primary tubercle in a man over twenty-five should contain living tubercle bacilli

The acute reactions obtained when a primary infection is administered to an animal with fully developed silicosis have anatomic counterparts in the so called "perinodular" type of human tuberculosis, a rare form which runs a rapid course with a fatal outcome. The cases usually occur in young subjects who appear to have little resistance to infection. From the animal experience it is assumed that they are nonimmune subjects whose first contacts with the tubercle bacillus were made after silicosis had developed

The chronic forms of the infection are more apt to occur in individuals possessing some resistance by virtue of previous infections at an earlier age. Such persons may enter employment involving an exposure to silica dust already reinfected and carrying either a healed or a latent but potentially active focus of reinfection in the upper portion of their lungs Inhaled silica accumulates in unusual concentrations in and about the scars of infection There its action accentuates the existing tendency to fibrosis and the scar becomes still thicker and heavier. If the focus is sterile and contains no living organisms more fibrosis is the only outcome If, on the other hand, viable tubercle bacilli are sequestered within areas of caseation it is altogether probable that in time the phagocytes will transport silica into them. When enough reaction has occurred the organisms will begin to multiply

again, as was observed in the experimental animals Here are the potentialities for spreading tuberculosis but unlike the focus in the animals, the human lesion is old, dense and often very thick The proliferating organisms spread into an environment of scar tissue with a deficient blood and lymph supply and a low oxygen tension Consequently the stimulating effect of the silica is more or less neutralized and the bacilli are barely able to maintain themselves. Enough remain alive so that in combination with the silica they continue to cause proliferative reaction The resultant area of silico tuberculosis slowly increases in size but the toxic symptoms of infection are slight or absent because the opportunities for absorption are limited Ordinary doses of tuberculin fail to elicit reactions in the skin, but larger amounts will produce them (This has been amply demonstrated in an unreported series of such tests by Dr L E Hamlin,[40] 1936 ) In time enough bacilli make their way to the surface of the involved area where they find more normal conditions for growth Then they multiply more rapidly, toxic products are absorbed and the ordinary clinical symptoms of infection are manifest. The bacilli, no longer bound in the scar tissue, reach the bronchi and the sputum may become positive for the first time Now the nature of the process becomes clinically obvious

Not all the chronic cases of silico-tuberculosis result from the reactivation of preexisting latent apical foci Individuals, resistant by virtue of previous infections, may become reinfected early in the course of their exposure to dust If conditions are such that the focus of reinfection is encapsulated and does not spread immediately, chronic disease may again develop Such perhaps are the lesions found in the middle or lower parts of the lung which are unusually common in silicotic subjects The evolution of this type of silico-tuberculosis is essentially the same as that just described Its coun-

terpart is not found in the animals for none of them have received exogenous reinfections. However, the principle of resistance acquired by virtue of previous non-progressive infection is illustrated in the animal whose primary infection remains quiescent for a time but when it is activated the resultant endogenous reinfection is of a chronic type.

There are also human cases of tuberculosis on a background of silicosis which behave as do those in the general population They generally occur when the exposure to silica has not been severe or prolonged One might assume that insufficient quantities of silica have established contact with the tuberculous focus to affect its behavior since in animals such contact seems to be the essential requirement.

The influence of inhaling other types of dust upon pulmonary infection with $R_1$ tubercle bacilli have already been mentioned Detailed discussion of their individual effects is unnecessary except to state that in general the results are comparable to clinical experience in industries where the dusts concerned are generated. One notable exception exists in the case of the carbide of silicon which in animals caused the infection to progress almost as regularly as free silica. This was true in several guinea pig experiments where the dust was inhaled (1923) and the infection was administered either by inhalation or by injection at remote points. It was not confirmed by the white mouse tests performed by Vorwald and Landau [20] (loc cit) An unusual incidence of tuberculosis has not been discovered in persons engaged in the manufacture of silicon carbide (Clark, 1929 [41]).

Perhaps the most striking feature of the experiments dealing with the combined action of different dusts and tuberculous infection is the variability of the response in the members of the different series While most of them exhibit a tendency to react in the same general manner, exceptional individuals behave atypically. The same observation is, of

course, constantly being made in human beings The result could hardly be otherwise when dealing with two living variables.

There remains for discussion the effect of the infection upon the pneumoconiosis It has already been stated that infection favors the development of nodule-like lesions in the case of granite dust It has also been intimated that some of the silicates may possibly become more active in the presence of infection It has been shown that infection is not essential to the formation of nodules by pure silica but that their development is accelerated when infection modifies the tissues of the host

The significance of the healed fibrous tuberculous lesions associated with the modified forms of silicosis needs special emphasis Anatomically the experimental lesions consist of wide areas of obliterating fibrosis whose evolution has been that of an organizing pneumonia In its earlier phases the various manifestations of tuberculous granulation tissue are obvious but later the specific evidences of the infection tend to disappear. In the tuberculous animal, exposed to ferruginous chert in particular, the healing is often so complete that the cause of the fibrosis would not be suspected without the evidence from previous members of the series The fact that human cases of sidero-silicosis have sometimes presented lesions which were obviously tuberculous in origin but in which living bacilli could not be demonstrated by guinea pig inoculation indicates that in them the causative organisms may die This suggests that at least some of the areas of massive fibrosis without histologic evidence of their etiology may also have originally been caused by tuberculous infection In several instances the position of the lesions in the upper portions of the lungs and their gross appearance have supported such a view Obviously more human autopsy material from persons exposed to many kinds of dust must be

studied before generalizations are possible. Nevertheless, it would now appear that many mineral dusts either alone or in combination with free silica, may favor ultimate healing of tuberculous infections with the formation of scar tissue from which all the specific characteristics have disappeared Confirmation of this hypothesis would be most useful in explaining the etiology of many of the areas of massive fibrosis found in human silicosis.

Attention is called to the fact that much of the material presented in this chapter has never been published. The writer has borrowed freely from the observations of his coworkers in the Saranac Laboratory. It is his pleasure to acknowledge his debt to Mr.D.E Cummings, Dr.A.J.Vorwald, Mr T M Durkan, Mr M Dworski and to all his other associates who are collaborating with him in the study of these problems. He also is indebted to the Journal of Industrial Hygiene and Toxicology, and the American Review of Tuberculosis for permission to reproduce numerous illustrations that have previously appeared in these journals.

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# VI. OCCUPATIONAL, PREVENTIVE AND LEGISLATIVE ASPECTS

## *GREAT BRITAIN*

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Silicosis as an industrial malady is probably of great antiquity in Great Britain. Collis[1] has quoted references to prehistoric factories where flint implements and weapons were made, and has found their modern counterpart in the workshops of Brandon, in Suffolk, where the few remaining flint-knappers make gun-flints for barter amongst the native races of Africa. In less remote times flints were used as building material, and, it may be presumed, the masons of the period suffered from the disease. "Stonemasons' phthisis" has been well known for generations amongst workers in sandstone, "grinders' rot" amongst metal grinders, "potters' asthma" amongst pottery workers, and "miners' phthisis" amongst metalliferous miners. These were all trade names for the same disease, silicosis or silicosis accompanied, as it frequently was, with tuberculosis.

The exact extent of the disease in modern industry is not accurately known, but there is an increasing tendency amongst medical men to make use of the term silicosis to indicate the disease in certificates of the cause of death, and from this source an approximate estimate can be made of the annual number of deaths occurring from this cause in England and Wales. During the seven years 1930 to 1936, the average number of deaths recorded in this way was 325 per annum. The principal occupations from which these fatal cases were derived are briefly described

## TABLE 1

| Industry | 1930 | 1931 | 1932 | 1933 | 1934 | 1935 | 1936 | Total |
|---|---|---|---|---|---|---|---|---|
| Sandblasting | 10 | 17 | 5 | 10 | 3 | 12 | 10 | 67 |
| Steel foundry work | 1 | 4 | 1 | 1 | — | 1 | 5 | 13 |
| Metal grinding | 35 | 47 | 21 | 21 | 18 | 28 | 24 | 194 |
| Metallurgist | — | 1 | — | — | — | — | — | 1 |
| Metal drilling | — | — | — | — | — | — | 1 | 1 |
| Stone, pebble flint and sand crushing | 1 | 2 | 2 | 4 | 1 | — | 1 | 10 |
| Railway construction (desert sand in Africa) | — | — | — | — | — | — | 1 | 1 |
| Refractories Industries | 11 | 19 | 5 | 7 | 9 | 12 | 9 | 72 |
| Abrasives Manufacture | — | 1 | 3 | — | 1 | 3 | 2 | 10 |
| Enamel Manufacture | — | — | — | — | 1 | — | 1 | 2 |
| Sandstone quarrying and dressing | 17 | 25 | 13 | 29 | 33 | 40 | 43 | 200 |
| Sandstone masons | 35 | 43 | 44 | 75 | 53 | 61 | 59 | 374 |
| Grave diggers | 1 | — | — | — | 2 | 1 | — | 4 |
| Tunnel mining (sewage works) | — | 1 | — | 3 | 1 | 3 | — | 8 |
| Granite quarrying and dressing | 2 | 1 | — | 3 | — | 1 | — | 7 |
| Slate quarrying and dressing | 1 | 2 | 5 | 3 | 3 | 3 | 6 | 23 |
| Slate dressing, with sand | — | — | 1 | — | — | — | — | 1 |
| Glazier & diamond setter | — | — | — | 1 | 1 | — | — | 1 |
| Pottery Manufacture | 53 | 57 | 61 | 53 | 44 | 58 | 49 | 376 |
| Leather dressing | 1 | — | — | — | — | — | — | 1 |
| Mining—Gold | 24 | 29 | 16 | 17 | 18 | 16 | 4 | 124 |
| Tin | 10 | 15 | 24 | 17 | 18 | 25 | 24 | 140 |
| Lead | 2 | 4 | 6 | 1 | — | 2 | 1 | 16 |
| Copper | — | 2 | 1 | — | — | — | — | 1 |
| Hematite | — | 10 | 7 | 6 | 13 | 12 | 9 | 57 |
| Coal | 41 | 50 | 76 | 74 | 85 | 111 | 120 | 557 |
| Fireclay | — | — | — | 1 | — | — | — | 1 |
| Barytes | — | 1 | — | 1 | 1 | 2 | 1 | 6 |
| Fluorspar | — | 1 | — | — | — | — | — | 1 |
| Mining engineers | 2 | 2 | 1 | 2 | 2 | — | 1 | 10 |
| Totals | 247 | 330 | 308 | 329 | 307 | 391 | 369 | 2 285 |

Annual average for the period of 7 years 325.86

### SANDBLASTING

This is the process of projecting sand, crushed flint or other grit, by means of compressed air, against a surface. It is used (1) in metal works, to remove adherent sand from castings and to produce a surface suitable for treatment by coating with enamel or another metal; (2) for etching glass and treating other non-metallic articles, such as wood and ivory. The process is carried on in many industrial centres throughout the country.

It can be carried out in various ways. For large metal castings the operator works in a specially constructed chamber, moving about and directing the sand-blast as required. The




FIGURE 1

A gun flint from Brandon Photo by S H Wilkes

atmosphere of the chamber is filled with a dense cloud of fine dust of the abrasive sand, which becomes finely comminuted in the process. The operator wears protective clothing and a helmet supplied with pure air under pressure through a tube Exhaust draught is provided for removing the dust-laden air from the chamber Smaller articles are treated in a closed cabinet before which the worker stands, passing his arms through guarded holes to direct the sand-blast, while watching the process through a glass panel A modification of this type of apparatus is that generally used for treating pottery For etching glass a stencil may be used, the glass being pressed against it while the stream of abrasive etches the pattern or

the exposed parts of the glass surface Other types of apparatus for treating metal articles are the turntable and revolving barrel in which the sand-blast is directed from one or more fixed points, and close attention by the worker is not necessary.

A more recent process is the use of the sand-blast for applying sand to the surface of bricks and tiles in the plastic state It is done in a special apparatus, the bricks or tiles passing on a conveyor band through a chamber in which they meet moving jets of sand, the sand adhering to the surface of the articles. Exhaust draught is applied within the chamber, and the openings are guarded with rubber or canvas flaps

The sand-blast has also been used to clean the outside walls of buildings and the hulls of ships

The abrasives in general use for sand blasting have been quartzose sand, which contains a high proportion of free silica , and crushed flint, a hydrated form of free silica Owing to the high incidence of severe silicosis amongst sand-blasters an increasing tendency has been evident, on the part of employers, to replace sand and flint by non-siliceous abrasives Instead of sand or flint, metal grit is being used for cleaning metal castings and surfacing metal for enamelling or metallisation , for glass, pottery and other articles alumina or other abrasives containing no free silica are used with satisfactory results.

With the use of quartz sand and flint, risks are incurred by workers through defects in the appliances or lack of maintenance, and by faulty or careless methods, particularly, opening the door of the cabinet or removing the helmet before the dust cloud has been removed by the exhaust draught

The dust hazard in cleaning castings is from the moulding sand and from the abrasive, when this is sand or flint Where clean castings or other metal articles are treated, the danger is from the abrasive alone When metal grit is used on cast-

ings with moulding sand still adhering, siliceous dust is produced from the moulding sand. With clean metal articles and metallic abrasive all danger of exposure to siliceous dust is obviated. Certain objections have been raised to the use of steel grit as the abrasive, owing to technical difficulties which have been met with; for example, particles of steel becoming embedded in castings of non-ferrous metals or alloys and preventing satisfactory machining afterwards; or iron staining from particles of the abrasives being left on articles which were later enamelled. Such difficulties have been got over by using non-ferrous and non-siliceous abrasives, or by cutting out the blasting operation altogether. The pressure of the compressed air is varied with the nature of the work; for metal castings it is from 30 to 60 pounds to the square inch, while for pottery it is from 15 to 30 pounds; and for glass, wood and other materials it is still lower The lower the pressure of the air blast the less dust is likely to be formed.

Both sexes are employed on sandblasting small articles, but men only on the heavier metal work. In the census of England and Wales, 1931, the total numbers over sixteen years of age employed on sandblasting are given as: males 1295, females 100. The number of deaths certified due to silicosis in England and Wales in the seven years, 1930 to 1936, was 67 or 9 6 per annum. This gives a mortality rate of 6 9 per 1000 living. The rate is actually higher than this, because the census figures include shot blasters working on clean metal, who are not exposed to siliceous dust.

Silicosis occurs amongst sand-blasters after short periods of employment, compared with other occupations. In 38 fatal cases which were fully investigated, employment was less than twenty years in all of them, in 8 it was less than five years, in 15 between six and ten years; in 11 between eleven and fifteen years, and in only 4 it was over sixteen years.

*Prevention.* Regulations for the Grinding of Metals (Mis-

cellaneous Industries) 1925[2] provide that (1) sand-blasting shall not be done in any room except in an enclosed chamber or cabinet in which no other work is ordinarily performed and at which efficient means are provided to prevent escape of dust outside of the chamber or cabinet, (2) no person may perform sand-blasting in the open air, or work within 30 feet of such, unless he is wearing a suitable protective helmet and gauntlets; during sand-blasting in a chamber overalls are also required, (3) the protective helmet must be provided with a supply of pure air, means of escape for exhaust air, and it must bear the mark of the person using it; (4) before being used by another person the helmet must be thoroughly disinfected These Regulations apply to metal works in which grinding of metals and cleaning of castings are done. In other factories and workshops the general provisions of the Factories Act[3] are applicable Section 4 provides that provision shall be made for rendering harmless, so far as practicable, all dust etc that may be injurious to health Section 47 requires means to be taken for preventing the inhalation of dust, and Section 48 requires exclusion of persons from workrooms in which siliceous dust is produced during intervals allowed for meals or rest

Even with improvements in sand-blasting apparatus the margin of safety is so narrow in this hazardous process that the workers cannot yet be said to be fully protected The only solution of the problem at present in sight is the abolition of siliceous abrasives in sandblasting and the thorough cleaning free from moulding sand of castings before shot-blasting.

*Compensation* The Various Industries (Silicosis) Scheme, 1931[4] provides for payment of compensation in cases of death or total or partial disablement of workmen employed in foundries and metal works on 'sandblasting of metals or articles of metal by means of compressed air and with the use of quartzose sand or crushed silica rock or flint "

### Grinding of Metals

The processes which cause silicosis in the grinding of metals are confined to those in which grindstones composed of sandstone are used In addition to actual grinding, certain incidental processes of dressing the surface of the grindstone are very important from the health point of view.

The industries in which metal grinding is important are (1) the cutlery and edge tool trades, and (2) machinery and general engineering works and foundries.

Grinding may be done wet or dry, and the metal being ground may be moved by hand or by mechanical power on a machine In wet grinding, the grindstone is mounted on a horizontal shaft and is made to revolve rapidly in a direction away from the grinder, except in some special kinds of grinding, such as scythe-grinding, when it revolves towards the grinder. Water is laid on from a tap or spray above the grindstone and is drained away beneath it. Formerly the lower part of the stone dipped into water in a trough in which grit, metal and dirt were allowed to collect. Wet grinding is also done by machines, especially in the manufacture of files, saws and machine-knives, when the metal being ground is fixed to a part of the machine which moves under or across the revolving grindstone

The processes of dressing the grinding surface of the grindstone are · 'racing', that is, trueing the surfaces of the grindstone before it is brought into use for the first time; and 'hacking' and 'rodding' or 'scaring', which are carried out frequently in the course of the day's work to maintain the grinding properties of the surface.

Grindstones are obtained chiefly from the millstone-grit sandstones of the Derbyshire, Sheffield and Newcastle districts They vary in size from 1 to 7 feet in diameter and from 1 to 12 inches in thickness

Dust is produced by attrition of the grindstone in all processes in the grinding of metals, and it varies in amount with the hardness of the stone, the shape of the article being ground and the amount of pressure used. Concentration of dust found at breathing level of the grinder in various processes in wet grinding are as follows (the principal figures being number of particles in 1 cubic centimetre of air, the figures in parentheses being estimated millions of particles in 1 cubic foot of air). (1) Hand-grinding, table-blades, 280 (8); chisels, 1150 (33), scythes, 660 (19) (2) Machine-grinding, paper-knives 130 (3.7), files, 270 (7.7), edge-tools, 120 (3.4) In dry grinding sheep-shears by hand (with exhaust draught), 240 (7).

The change from grindstones, made from sandstone, to artificial abrasive wheels, made from carbide of silicon and various forms of alumina, has been very marked since 1927. It was estimated that in 1926 about 1600 grindstones were in use for hand-grinding in the Sheffield district This number was reduced to about 200 in 1933 and many of these were not in regular use In the Birmingham district there were 480 grindstones in use in 1926 and 452 persons employed In 1936 only 22 of those sandstone wheels were in use and 18 persons were employed

Owing to this rapid and extensive change in the method of working it is not possible to estimate the numbers of persons on whom the present incidence of silicosis is falling It must be presumed that the deaths in these later years are the result of conditions which existed between twenty and forty years ago The numbers of deaths certified to be due to silicosis amongst metal grinders is showing a tendency to fall In the seven years, 1930–36 the annual figures were 35, 37, 31, 21, 18, 28 and 21, average 27 7 per annum This gradual fall in incidence of silicosis is chiefly due to the change from sandstone to abrasive wheels At the coming into force

of the Workmen's Compensation (*Silicosis*) Scheme for the grinding of metals industry on 1st July, 1927, there was a widespread departure from the use of sandstone wheels and a corresponding adoption of artificial abrasives to which the Compensation Scheme did not apply.

*Prevention* Another influence in reducing silicosis in this industry is the stringent preventive measures enacted in two codes of Regulations in 1925 : the Grinding of Metals (Miscellaneous Industries) Regulations,[5] and the Grinding of Cutlery and Edge Tools Regulations.[6] These two codes replaced a single code of Regulations made in 1909, and followed a technical and medical inquiry[7] in the industry. The general provisions of the Regulations may be summarised thus —

(1) *Localised exhaust draught for 'racing,' dry-grinding or glazing*
(2) Separation of the process of 'racing' grindstones in the cutlery and edge tool trade, and of wet grinding
(3) General ventilation sufficient to provide 12 changes of air per hour, and 15 changes in cutlery and edge tool shops, and continuous movement of air in a direction from the grinder to *the outlets*
(4) Suppression of dust at 'hacking' and 'rodding' by water or other means
(5) The rooms must be a certain minimum height, windows must be adequate and kept clean, floors and walls must be so constructed as to be capable of being cleaned, and all shafts, belts, etc efficiently covered
(6) Adequate drainage for water.
(7) Periodical cleaning of floors, walls, ceiling and fixtures
(8) Spitting prohibited
(9) Cloakroom accommodation to be provided
(10) Grindstones must not be less than the prescribed distance apart

*Compensation* The Metal Grinding Industries (Silicosi Scheme, 1931,[8] made under the Workmen's Compensatio Act 1925, and the Workmen's Compensation (Silicosis an Asbestosis) Act, 1930, replaces a Scheme made in 1927 It provides for compensation in cases of death or total or partial disablement certified to be due to silicosis or silicosis accompanied by tuberculosis, in workmen employed in

(i) grinding of metals (by means of a grindstone),
(ii) any work incidental to such grinding, and glazing, done in the same room as grinding,
(iii) racing of grindstones for the grinding of metals

## Refractories Industries

For purposes of legislation it has been necessary to define this industrial group, as meaning the following processes carried on at mines, quarries, factories and workshops at which refractory material containing not less than 80 per cent total silica is got or manipulated with a view to manufacture or sale, namely:—

All processes in or in connection with the getting, handling, moving, breaking, crushing, grinding and sieving of such material, and all processes in the manipulation of such material in the manufacture of bricks or other articles containing not less than 80 per cent total silica The processes include the quarrying or mining of the raw materials, usually sandstones of the coal measures (called ganister), which occur in open quarries and in mines, in some localities, sands or 'pocket-clays' are used They contain usually from 92 to 98 per cent of silica The material is crushed, mixed with suitable bonding substances, and made into silica bricks, silica cement, steel-moulders' composition, and similar products for use as refractories in the manufacture of metals, especially steel and steel-castings, and in the construction of gas-retorts and flues

Quarrying and mining of the solid rock, drilling, blasting and breaking it with hammers are processes attended with exposure to silica dust. Dust is also produced in crushing, grinding, sieving and mixing the material and in moulding bricks. Moving the bricks to and from the drying floors and setting them in and withdrawing them from the kilns in which they are fired also cause evolution of dust. The character of the dust varies with the composition of the raw materials. When these contain clayey matter the finest particles tend to form aggregates, thus reducing the risk. No process in this industry, however, appears to be entirely free from danger.

About 3,000 persons, including a small proportion of women, are employed in the industry, which is distributed in the coal measure districts, especially in the north of England, South Wales and central Scotland.

During the seven years, 1930–1936, the numbers of deaths from silicosis in the industry show a yearly average of 10, with little tendency to fall. Most of the deaths occur amongst ganister miners and quarrymen.

*Prevention* In the factory processes there is an increasing use of mechanical methods, and these will have the effect of reducing exposure to dust, particularly in the use of kilns which are not entered by the workmen

Refractory Materials Regulations 1931 [9] are a revised form of the code which was in force in factories since 1919. They require the enclosure and provision of exhaust draught for machinery were dusty material is crushed, sieved or moved, the use of water for suppression of dust, and the removal and disposal of debris from floors and passages.

Section 78 of the Coal Mines Act, 1911, requires generally that mechanical drills used for drilling in highly siliceous rocks shall be provided and used with an efficient means to prevent the escape of dust into the air. Under the same Act

general Regulations have been made for ganister mines, and under the Metalliferous Mines and Quarries Acts, special rules have been established in tin mines and in many quarries where ganister, sandstone and other silica rocks are worked

*Compensation.* The first scheme of compensation for silicosis was made for the Refractories Industries under the Workmen's Compensation (Silicosis) Act, 1918, and it came into force on February 1, 1919 It provided for initial and periodical medical examination of all workers, suspension from the industry on a diagnosis of simple silicosis, as well as of silicosis with tuberculosis, and compensation in respect of disablement or death from silicosis or silicosis with tuberculosis

The Scheme was revised in 1925, following an Inquiry by a Departmental Committee, and again in 1931 (11) to provide for examinations being made by a Medical Board

## MANUFACTURE OF ABRASIVE SOAP POWDERS

These products are used chiefly as domestic abrasive cleansers They consist of ground silica, pumice or similar abrasive substance, mixed with smaller quantities of anhydrous sodium carbonate (soda ash) and powdered soap, and sometimes other substances

Except in a few of the largest works the abrasive mineral is obtained already finely ground When silica is used it is generally a highly siliceous quartzite rock containing 96 to 98 per cent of silica, and it may be calcined before it is ground The silica 'flour', as it is called, is mixed with the soda ash, soap, etc, either by hand with a shovel, or in a mixing machine. The cartons may be filled by hand, or the mixed powders are conveyed and filled mechanically

A few years ago a considerable number of small undertakings were started, sometimes as a branch of another business, the proprietors and workers having little or no knowledge of

the risk to health or the means necessary for avoiding it. The result has been that some of the most rapidly developing cases of silicosis on record have occurred before adequate measures could be taken to deal with the danger.

In all the fatal cases of silicosis recorded in England from the manufacture of these abrasive powders, the dust has contained a highly siliceous quartzite rock, finely powdered and mixed with soda ash and soap powder The number of deaths certified due to silicosis contracted in this manufacture was 10 during the seven years 1930–36. This is probably less than the actual number In one factory where the manufacture was carried on to a substantial extent for seven years, 13 persons are known to have died from silicosis with or without tuberculosis, out of a total of 59 persons who had been employed during that period for longer than two months on the process All except one had commenced the work between fourteen and sixteen years of age.[11] The unusual characters of the rapid onset and course of the disease in most of these cases may be connected with the early age of the subjects. Attention has been drawn to the presence of free alkali in the powders to account for these characters.

Mention may be made here of siliceous materials used in polishing compositions The best known are Tripoli, Neuburg chalk and Diatomite.

*Tripoli* is regarded as a chalcedonic form of silica containing 96–99 per cent silica. The substance generally known by the name is obtained from Seneca, Missouri The name is derived from Tripoli in North Africa, where the product is diatomite.

Two fatal cases of silicosis with tuberculosis have been recorded from exposure to dust of tripoli The two men had been employed for ten and eleven years respectively mixing the ingredients in the manufacture of a foundry sand in one case, and of polishing-composition in the other case.

*Neuburg chalk* is a natural deposit of siliceous material at Neuburg on the Danube The silica content is variously given as from 65 to 97 per cent Definite cases of pulmonary disease have occurred from exposure to the dust of Neuburg chalk in Britain and in Germany

*Diatomite* (kieselguhr, diatomaceous earth) is formed from the siliceous skeletons of minute aquatic plants and when pure it contains up to 96 per cent of silica It is used as a polishing medium, as filtering material and for its insulating properties Radiological examination of 6 workers exposed for ten years to the dust of kieselguhr in Britain showed evidence of a slight degree of fibrosis in 2 of them and none in the others [12]

*Prevention.* Suppression of all dust in the processes of mixing and packing of abrasive soap powders is difficult to attain, even with the use of exhaust draught, owing to the dryness and fineness of the powders Reliance is best placed on the fullest use of mechanical methods, enclosure of the machinery and the application of exhaust draught so as to maintain an inward directional air current at all points where feeding or discharging takes place. In some factories alternation of occupations is practised, and in a few cases periodical medical examinations are made with a view to suspension on the earliest sign of respiratory trouble

*Compensation* The Various Industries (Silicosis) Scheme, 1931 [4] applies to the "breaking, crushing, grinding, sieving, mixing or packing of silica rock, or of dried quartzose sand or any dry deposit or dry residue of silica or any dry admixture containing such materials, or any process ancillary thereto" The Scheme provides for compensation in respect of workmen certified to be suffering from silicosis or silicosis accompanied by tuberculosis to a dangerous or disabling extent, or to have died from the disease

### The Sandstone Industry

This industry comprises all processes in getting sandstone from a quarry or mine, and in cutting and dressing it.

The sandstones include the sedimentary silica rocks, and they are chiefly used in the construction of buildings, bridges, reservoirs and roads, and in the manufacture of grindstones and pulpstones. The industry is principally carried on in the districts where the quarries are situated, but stone-dressing takes place to some extent over the whole country, and every town has its stonemasons' yards. About 12,000 men are employed in the processes in quarries and, in addition, about an equal number of masons are employed in builders' and sculptors' yards

The workers include rock-getters who work at the stone face in quarry or mine ; quarrymen or stone-cutters who hew the blocks ; masons who shape and carve the stone to dimensions or patterns , drillers with hand or pneumatic-tools or steam-drills ; crushermen , labourers and cranemen ; and builders "fixers or "wallers", who frequently do some dressing of the stone.

The siliceous sandstones consist essentially of quartz grains, mixed with a variety of other minerals and held together by a cement of varying composition and proportion. The proportion of silica in sandstone varies in amount up to over 99 per cent.

All the processes give rise to dust, and the risk of silicosis depends on the concentration at breathing level. The following are average concentrations of dust found at breathing level in different classes of work in the sandstone industry ( the higher numbers indicate number of particles in 1 c c of air, the numbers in parentheses are calculated as millions of particles in a cubic foot) : masons, 545 ( 16 ) ; rock-getters, 319 ( 9 ) ; drillers, 939 ( 27 ) ; quarrymen, 1956 ( 56 ) ;

crushermen, 1132 (32). Factors which appear to influence the concentration were determined by Green and Watson.[13]

'The highest concentrations of dust are obtained when the mason is punching or roughing The following figures show the order of the number of particles produced:

2,150 per c c mason in closed yard, almost dead calm
1,322 per c c. mason in open yard on threshold of shed, almost dead calm
537 per c c mason in open yard, almost dead calm
313 per c c mason in closed yard, strong draught
396 per c c mason in open yard, gusty wind

'It would be difficult to assess the average exposure of a mason over a long period of time Even though 40 samples were taken each for periods of one hour upwards, it has not been possible to obtain sufficient data to yield a value on which any reliance can be placed Extreme values can be stated, viz, 2,150 per c c to about 200 per c c for the closed yard and 1,300 per c c to 150 per c c for the open yard

'In contrast to the large variations in numbers, the size-frequency figures show a remarkable degree of constancy They emphasize the preponderance of the small particle and the relative infrequency of particles over 5 μ in diameter'

In an investigation into the occurrence of silicosis in the sandstone industry, Sutherland and Bryson[14] found the disease amongst masons, rock-getters, quarrymen, planers and wallstone dressers Roentgenological evidence of the disease was most frequently found after forty years of age and after twenty years in the industry Ferguson[15] found clinical evidence of pulmonary fibrosis in 133 sandstone workers out of 1,000 consecutive routine examinations of men continuing at work Roentgenographic examination of 173 of the most definite of these showed silicosis in 109

During the seven years 1930–1936 the number of deaths certified due to silicosis amongst sandstone workers in Eng-

land and Wales was 574, the annual average being 82 deaths Estimating the number employed at 25,000 this gives an annual death-rate of 3 3 per 1000. The numbers for the consecutive years over the period show no tendency to fall, the last four years being above the average

*Prevention.* In processes carried on in quarries in which the stone contains not less than 80 per cent. of silica, Special Rules made under the Quarries Act, 1894, are applied by the Mines Department. The Special Rules provide that: —

(1) Rock drilling by mechanical power is done with an efficient water jet, or other efficient means to prevent escape of dust

(2) Stone breaking by manual labour is to be done only in the open air, and respirators provided if workers are exposed to the inhalation of dust

(3) Machines for crushing and grinding of stone to be (a) provided with exhaust draught and dust collecting appliances, or sufficient water or steam spray, or other arrangement to prevent escape of dust, or (b) entirely enclosed

(4) Stone to be kept wet in sawing, planing and turning

(5) Workmen dressing stone not to work nearer than 6 feet from each other

(6) Stone dressing not to be done in any closed shed unless exemption is granted on the ground of exhaust ventilation being provided

The stone mason who dresses the stone by hand is the most frequently affected in the industry, and preventive measures are neither easily applied to this work nor readily adopted by the masons themselves Progress has been made in the development of an efficient type respirator which will protect against the finest dust to the extent of 80 per cent while affording a reasonable degree of comfort, at least for intermittent wear. If such respirators were worn regularly during

certain phases of the work which have been shown to be most productive of dust, many lives would be prolonged Two methods are available for applying localised exhaust draught to the process of hand dressing either with hammer and chisel or with a pneumatic tool. In one of these a flattened tube of gutta-percha is fixed to the back of a glove or mitten worn on the left hand, and this is connected with a flexible tube leading to an injector arrangement on a compressed air supply. In the other a small metal hood connected with a flexible duct is placed as near as possible to the chisel point and is moved about as the work progresses The tube is connected with a dust-collecting appliance

*Compensation* The Sandstone Industry (Silicosis) Scheme, 1931[16] applies to workmen employed in the "Sandstone Industry", defined as follows, —all processes in or incidental to the getting or manipulation of sandstone with a view to manufacture, sale, or use, which are carried on at or within the close or curtilage of any mine or quarry or at any premises worked in conjunction with a mine or quarry, wherever situate.

Exemptions from this Scheme are provided for places to which the Refractories Industries Scheme applies, premises not being a mine or quarry for the making of silica flour, any mine or quarry worked occasionally only and where no explosive or mechanical power is used in the processes, workmen employed exclusively as craneman, engineman fitter or blacksmith, manufacture of artificial stone except as regards breaking, crushing or grinding sandstone

The financial and administrative structure of this Scheme resembles that for the Refractories Industry, in that all claims for compensation and all expenses under the Scheme are paid out of a General Compensation Fund maintained by means of levies payable by all employers in the Industry The Fund is managed by a Company representative of the Employers,



FIGURE 2 See caption on page 365.

but cannot be wound up without the Secretary of State's approval all levies have to be approved by the Secretary of State. A large proportion of the workmen engaged in the processes of dressing or manipulating sandstone, e.g. building and monumental masons, are not covered by this Scheme because their employers, not being owners of a mine or quarry, are not contributors to the funds of the Company All such workmen are covered by The Various Industries (Silicosis) Scheme 1931 [4]

The Silicosis and Asbestosis (Medical Arrangements) Scheme 1931,[26] and the Scheme as amended in 1934 [27] provides, with certain exceptions, for initial and periodical medical examinations of workmen employed in prescribed processes in the industry The intervals between periodical examinations are eighteen months in the more hazardous processes, and three years in those in which the risk of contracting silicosis is considered to be less. The periodical examinations are made by the Medical Board, and in most cases at the place of employment.

### Granite Industry

The granites are igneous rocks of crystalline structure, in which the amount of free silica as quartz rarely exceeds 1/3 of the total, and in some granitic igneous rocks frequently included in the commercial term, the proportion of free silica is much less than 1/3, the other constituents being silicates The granites and allied rocks occur chiefly in the Counties of Cornwall, Devon, Leicester, Cumberland and Westmorland, and in several parts of the west and north of Scotland.

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Figure 2

Localised exhaust for dust removal at stone-dressing The expanded end of the duct is made of rubber and is attached to the back of a glove worn by the operator By kind permission of the Home Office Industrial Museum



FIGURE 3 See caption on page 367.

The processes are carried on for the most part at or near the places where the quarries are situated. In getting and dressing the rock the processes resemble those in the sandstone industry, but owing to the greater hardness or toughness of the granites, pneumatic tools are much more frequently used, and one of these, the surfacing machine known as the 'dunter', is not met with in sandstone work. Some of the dressing of granite for monumental and architectural purposes is carried on in partly enclosed sheds, but generally most of the work is done in open sheds or in the open air. Workmen who have worked both in Britain and America believe that the risk is greatly increased in the latter country with the use of closed sheds made necessary by severe weather. Owing to the qualities of the granitic rocks they are largely used for road material and for concrete, and almost every granite quarry has its crushing plant, while in some quarries the whole output is in the form of setts, kerbs, and other road material.

Dust is produced in the various classes of work in granite. Average concentrations of dust found, expressed as particles in 1 c.c. of air (and in parentheses millions of particles in a cubic foot of air) were: masons' work 112 (12); rock getting 343 (10); drilling 1935 (55); stone-crushing 1831 (52).

In the roentgenograms of granite workers the prevalent type of shadows indicating fibrosis of the lungs is somewhat different from that found in workers who have been exposed to dust containing a high proportion of free silica. It is a finer type of mottling, with more linear striation and reticulation, but nodulation of silicotic type is also met with in some cases.

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FIGURE 8

Showing attachment of the expanded end of the duct and its connection with the suction apparatus. By kind permission of the Home Office Industrial Museum.

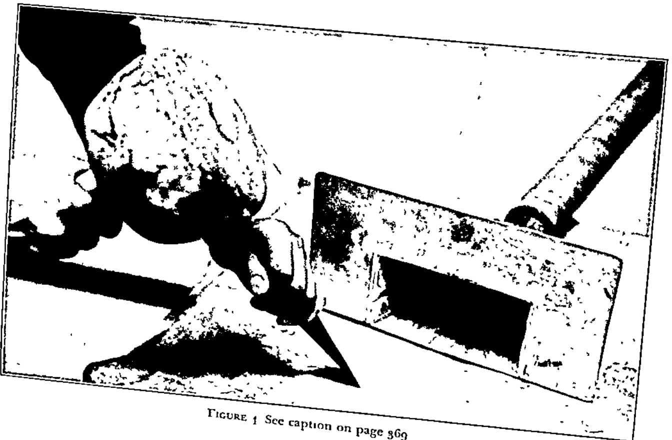


FIGURE 1 See caption on page 369

Pulmonary fibrosis of granite workers in this country is neither so prevalent nor so severe as amongst sandstone workers, and the same features have been noted in other European countries by Teleky, Gutzeit and others

An investigation was made by Sutherland and others,[17] in which 491 workers were clinically examined and 211 of these were examined roentgenologically Clinical evidence of fibrosis of the lungs was found in 260 cases (52.6 per cent), and roentgenological evidence of silicosis was found in 36 (17 per cent) of these Comparing the results of the medical examinations made by these workers in the granite and sandstone industries, the number of cases of fibrosis is seen to be of the same order, 52 6 and 59 per cent respectively Of those examined by roentgen-ray in the two industries, only 17 per cent of granite workers showed evidence of silicosis, compared with 49 per cent in the sandstone industry[14] During the seven years, 1930–1936, 7 deaths were certified due to silicosis amongst granite workers in England and Wales, and 1 in Scotland

*Prevention* The processes in the granite industry are similar to those in the sandstone industry, and the same methods of dust suppression can be employed in both The dust from ...uling can be dealt with by the application of water or by the use of a dust trap with exhaust For cutting, turning and surfacing, specially adapted methods of application of exhaust draught have been devised

*Compensation* The Various Industries (Silicosis) Scheme, 1931[4] provides for the payment of compensation only in the case of workmen employed in one specified process in the getting and manipulation of granite (including any igneous rock), namely 'the dressing of stone by masons

FIGURE 4

Movable hood connected with exhaust apparatus by a flexible tube By kind permission of the Home Office Industrial Museum

### Slate quarrying and Dressing

The industry is concerned with quarrying or mining the rock, and making and shaping the slates for roofing and other structural work Powdered slate is used to some extent in the manufacture of roofing felt for road material and as a 'filler.'

The industry is widely distributed in this country, but about 9,000 of the 11,000 wage earners in the industry are employed in North-West Wales.

In the open quarries of North Wales the rock is quarried by blasting and wedging. The large blocks are conveyed to the mills where they are sawn to sizes with power-driven saws cutting across the grain. The blocks are then split with chisel and mallet and cut to required sizes by hand or machine. In some small quarries there are no mills and splitting is done in the open air. Slate makers, who do the splitting and cutting, usually number 3 or 4 times as many as the rock getters.

Slate is the typical cleaved rock. Its most important constituent quantitatively is silica, free as quartz and combined as silicates of aluminum, iron, alkalis and other bases. In Penrhyn (Welsh) slates quartz amounts to 34 66 to 43 26 per cent, while total silica (free and combined) is 57.75 to 63.01 per cent [18]

Dust is produced in drilling in the quarry· the average of a series of samples taken with Owens' Jet Dust-counter showed a concentration of 560 particles in 1 c.c. of air (16,000,000 in 1 cubic foot) In the mills the concentration of dust varied widely, in a series of samples, from 241 particles in 1 c c (7,000,000 in a cubic foot), to 3,325 in 1 c c. (99,000,000 in a cubic foot); the average count was 1,463 in 1 c c (42,000,000 in a cubic foot).

Sutherland and Bryson [19] made clinical examinations of 120 workers and found severe fibrosis of the lungs amongst

the older millmen 3 of the 14 men with fibrosis had worked between thirty and thirty-nine years in the industry Radiological examination of 52 millmen showed signs of silicosis in 14 men. The rockmen were found to be less severely affected, while fibrosis was found amongst the older rockmen, no silicosis was found, although the average age of the rockmen (52 5) was higher than that of the millmen (46 1) During the seven years 1930–36, 23 deaths were certified due to silicosis amongst slate quarrymen and dressers

In view of the ages reached by many of the workmen while still remaining fit for work, it may be assumed that there is no general disability occurring amongst these workmen from exposure to dust in their occupation

*Prevention* The risk of injury to health in open slate quarries is not great In confined spaces in quarries and in slate-mines water may be used to suppress dust in drilling In the mills dust can be controlled by means of localised draught, or where this is not practicable, by a high standard of general ventilation, and sawing can often be done with copious application of water

*Compensation* The slate quarrying and dressing industry is not specifically covered by the Workmen's Compensation (Silicosis) Acts

## POTTERY INDUSTRY

This industry includes several distinct branches, sub division being determined by the nature of the article being manufactured and the materials entering into the composition of the ware The principal divisions are earthenware, which includes general earthenware, tiles, sanitary earthenware and electrical fittings, china, jet-and-Rockingham, sanitary fireclay, stone-ware, coarse ware The occurrence of silicosis is especially associated with the first two groups, namely, earthenware and china The great centre of the in

dustry is at Stoke-on-Trent in North Staffordshire, where all branches of pottery manufacture are represented.

In the manufacture of earthenware, the ingredients, — ball clay, china clay, china stone, and flint — in a ground state, are mixed together in the form of liquid slip, from which excess water is removed by pressing. The resulting plastic material is used by throwers, pressers, jiggerers and jolliers, handlers and modellers, in the potters' shops Increasing use is made of the method of 'casting' ware. For this process the slip or body is made into a liquid, which is allowed to flow by gravity from a reservoir, or is poured from a vessel by hand, into moulds made of plaster-of-Paris. After a period, to allow the porous mould to draw liquid from the slip, the excess slip is poured off, the mould is separated into its component parts, and the article removed for fettling, drying and finally, firing For the making of earthenware tiles and electrical fittings by pressure, the slip is made into a semi-dry powder, called "dust," which is pressed in moulds So long as the clay remains moist it is harmless, but in the process of manufacture fragments fall on benches and floors or adhere to the clothing of the workers, become dry, and give rise to dust After the article has been made in the plastic form, partly dried, it is frequently subjected to the processes of towing on a revolving disc, and fettling by hand, to remove irregularities of surface and edges. The articles are then placed in fireclay saggers with some sand, and fired in the oven. They are then termed "biscuit ware." Subsequent processes of decoration and glazing do not give rise to silicosis ; but, after the ware has been decorated and glazed, blemishes are removed by polishers who frequently use powdered flint, mixed with water, as a finishing abrasive on a rapidly revolving wheel.

In the manufacture of china, the ingredients are calcined bone, china clay and china stone, but it contains no added

flint The processes of making the articles are similar to those employed with earthenware in a general way, but in the process of firing the articles require much more support than do earthenware articles, and to provide this they are placed in saggers with finely powdered flint When the saggers of china ware are taken from the oven the flint which was used in placing them is removed by a series of processes known as "scouring"

For use in the manufacture of pottery, flint is calcined by burning the flint pebbles with coal fuel in kilns The calcined flints are washed or sieved to free them from ashes, and crushed in a stone crushing machine If the flint is required for making slip for earthenware it is then ground with much water in buhr stone mills This process is frequently carried on at the pottery where the flint is to be used If the flint is required for placing china biscuit ware it is reduced to fine powder, in the dry state, in crushing mills

It will be seen that the exposure to flint dust in the pottery industry, in addition to the preliminary processes of the preparation of the flint by milling and grinding and the final process of polishing, occurs amongst those manipulating the earthenware body in the plastic and semi-dry states, and, in the manufacture of china, amongst those employed in placing the biscuit ware and in removing the flint from it after firing It is in those processes that the highest incidence of silicosis occurs.

Estimations of atmospheric dust were made by H H Watson [20] with the thermal precipitator at various processes in pottery manufacture The following are typical examples of the concentrations found at breathing level, unless otherwise stated, and expressed in the number of particles in 1 c c of air (and, in parentheses, millions in a cubic ft of air) — flint-grinding, dry with exhaust draught 150 (13), flint milling, wet (general air of the room) 109 (3), placing

china biscuit ware (general air of the room), 121 (3.4); earthenware plate-making: 176 (5); fettling, damp: 168 (4.8); *fettling*, dry: 201 (5.7); pressing tiles: 213 (6); fettling tiles: 275 (8); casting sanitary earthenware (general air of the shop), 164 (4.7).

In a clinical and Roentgenological investigation in various occupations in the pottery industry, Sutherland and Bryson[21] found that of 182 males, 76 showed roentgenological evidence of silicosis, and of 68 females, 11 showed such evidence. Silicosis was diagnosed in flint millers, workers in the earthenware sliphouse and potters' shops, and in china biscuit placers, oddmen and scourers, and in polishers. Their findings were confirmed later by mortality experience in the industry. The figures for the seven years, 1930 to 1936, are given in the Table, together with the mortality rates

## TABLE 2

Pottery Industry. Mortality rates for silicosis in certain occupations calculated on fatal cases investigated by the Factory Department in the seven years 1930 to 1936.

| Occupation. | Estimated numbers employed | | Deaths in 7 years | | Annual mortality rates per 1000 employed. | |
|---|---|---|---|---|---|---|
| | Males | Females | Males | Females | Males | Females |
| Flint millers and mill labourers | 240 | – | 9 | – | 5 4 | – |
| Earthenware—sliphouse | 698 | – | 19 | – | 3 9 | – |
| " throwers | 24 | – | 1 | – | – | – |
| " turners | 132 | – | 3 | – | 3 3 | – |
| handlers | 113 | 336 | 4 | – | 7 1 | – |
| " platemakers | 364 | 308 | 19 | 1 | 7 5 | 1 3 |
| " dishmakers | 113 | 6 | 7 | – | 8 8 | – |
| " saucermakers | 52 | 156 | 4 | 1 | 11 0 | 1 3 |
| " cupmakers | 46 | 248 | 2 | 5 | 6 2 | 2 9 |
| " jolliers | 201 | 122 | 25 | – | [illegible] | – |
| pressers and casters | 221 | 592 | 29 | 1 | 18 7 | 0 4 |
| " (sanitary) " " " | 736 | – | 39 | – | 7 6 | – |
| " (tiles) " " " | 229 | 492 | 9 | 6 | 5 7 | 1 7 |
| hollow-ware pressers | 248 | – | 30 | – | 17 3 | – |
| China biscuit ware placers | 152 | – | 18 | – | 16 4 | – |
| " odd men | 70 | – | 8 | – | 15 7 | – |
| " " scourers | – | 223 | – | 8 | – | 4 9 |
| Polishers | 330 | – | 21 | – | 9 1 | – |
| Totals | 4,029 | 2,283 | 247 | 22 | 8 8 | 1 4 |

calculated on the estimated number of persons employed in each occupation.

*Prevention.* Regulations were made in 1913, under the Factory and Workshop Act, for the Manufacture and Decoration of Pottery.[22] A Departmental Committee on Compensation for Silicosis dealing with the Pottery Industry reported on the working of the Regulations in 1928[23] and, following their recommendations, another Code, The Pottery (Silicosis) Regulations, 1932[24] was made. This Code deals entirely with the risk from the inhalation of siliceous dust, and it covers not only potteries but also factories in which flint is ground for use in the manufacture of pottery. The two Codes together provide a comprehensive measure for the control of dust in the industry. Special use is made of localised exhaust draught at specified processes, unless the material is so damp that dust is not produced; conveying machinery must be enclosed as well as being provided with exhaust draught; dust discharged from the exhaust fans must be collected in suitable apparatus; floors and benches must be freed from scraps and debris and cleaned daily, by a moist method after working hours, or by vacuum cleaner, and floors must be kept in good repair and clear of obstacles so as to facilitate thorough cleaning. The employer must appoint one or more competent persons whose duty it shall be to carry out systematic inspection of the working of all the Regulations, and to record any breach of the Regulations or any failure of the apparatus needed for carrying out the provisions, and the steps taken to remedy the defects or to prevent recurrence.

With a view to removing the danger from the use of powdered flint for firing china ware and the exposure to flint dust in removing it after firing, manufacturers experimented with various substances in the search for a safe substitute for flint. They found that sufficiently satisfactory results were obtained with alumina, and the Medical Research Council were

eventually asked to determine whether there would be any risk of pneumoconiosis from exposure to alumina dust. As no satisfactory data on the effects of alumina were available an investigation [25] was carried out on 50 workers who had been exposed to dust of alumina for periods varying from five to forty years at the furnaces in aluminium works As a result of clinical and radiological examinations of these men, and a statistical report on sickness rates amongst a larger group of similar workers, no evidence was found that alumina dust had caused fibrosis of the lungs Following this finding it seems probable that alumina will be used to replace powdered flint for the placing of china biscuit ware, and thereby the risk to china placers, oddmen and scourers will be removed. Alumina is also being used instead of flint as an abrasive for polishing finished ware.

*Compensation* The Silicosis and Asbestosis (Medical Arrangements) Scheme, 1931 [26] and the Scheme as amended in 1934,[27] made under the Workmen's Compensation (Silicosis and Asbestosis) Acts, provide, with certain exceptions, for initial examination, within two months of employment in the industry, and periodical examinations at prescribed intervals of persons employed in certain processes in the pottery industry, which may be summarised as *follows*.— (1) milling and grinding of flint or quartzose material; (2) in the manufacture of china, placing the ware in flint for firing and removing the flint after firing; (3) male workers in certain processes, carried on in potters' shops, in the manufacture of general earthenware, sanitary earthenware and earthenware tiles, and (4) polishers who use flint or quartz.

The Various Industries (Silicosis) Scheme 1931 [4] provides for the payment of compensation to workmen employed in the following processes and *potteries* · — (a) the milling of flint or crushing or grinding of silica rock or dried quartzose

sand ; ( b ) any process in or incidental to the manufacture of china or earthenware including sanitary earthenware, electrical earthenware or earthenware tiles, up to and including the preparation for glazing but excluding underglaze decorating, and modelling and mould making where these processes are carried on in separate rooms , ( c ) polishing, sorting or grinding on a power driven wheel in connection with the grinding of glost ware, and tile-slabbing

## Tin Mining

In this country tin is mined almost entirely in Cornwall The numbers employed vary with the economic condition of the industry , thus in 1928 there were 2,800 wage earners, and in 1934, 1,615

The ore occurs in lodes which traverse granite, or an argillaceous schist called 'Killas ' Dust is derived from the lodes which consist of hard stone ( containing the binoxide of tin ) , and from the containing rock of granite or Killas

In the Report on the Health of Cornish Miners [28] the high mortality from respiratory disease is referred to Out of 142 deaths of rock drillers, 120 were certified as from various forms of phthisis or "miners' disease," and 13 from other respiratory diseases. The majority of these rock drillers had worked in other countries, 90 of them in the Transvaal but 38 of them had worked only in Cornwall

During the seven years, 1930 to 1936, silicosis was certified as the cause of death of 140 tin-miners, a mortality rate of 12 per 1,000 on the number of wage earners in 1934 It is not known how many of these tin-miners had worked in other mines, though no mention was made of such on the certificates

*Compensation* The Various Industries ( Silicosis ) Scheme 1931 [4] applies to all workmen employed *underground* in Tin

Mines, and in the breaking or crushing of the ore or the containing rock above-ground or any handling or moving incidental thereto.

## Hematite Iron-ore Mining

The industry is concerned with the mining of the anhydrous ferric oxide, haematite, $Fe_2O_3$. The numbers employed vary widely with the economic state of the industry, and during the present century fluctuations between 10,000 and 3,000 have taken place. The great majority of these are employed in the West Cumberland and North Lancashire iron-ore field, where the ore is used for metallurgical purposes, small numbers are employed in the haematite deposits in the South West, the product of which is used in the manufacture of paints

Much of the hematite deposits are precipitates from ferruginous solutions which must have been in most cases charged with $CO_2$, and such solutions would carry dissolved silica which would be simultaneously precipitated with iron, generally in the form of finely divided quartz. Also in cases where the calcium carbonate of calcareous rocks has been replaced by iron compounds, all the insoluble siliceous constituents of the original calcareous rock will find their place in the newly formed iron ore. Further, quartzose rocks may form one side of a lode of ore and these have often to be blasted or cut in order to work the ore-deposit Insoluble residue of the better class ores amounts to 5 per cent to 7 per cent, but rises to 15 per cent, mostly in the form of free silica In lower grade ores the insoluble residue may reach a much higher figure. Examination of thin sections of hematite ores reveals grains of quartz secondary silica and oxides of titanium, in addition to the various oxides of iron and carbonates The dust produced by drilling and blasting in haematite mines, therefore, will contain silica [29] A series of samples of dust from mine work-

ings, yielded on analysis from 2 76 per cent to 7 0 per cent free silica, and drillings from the ore body yielded 1 5 per cent to 6 8 per cent

H.H. Watson [30] found the following dust concentrations in a hematite mine at a distance of 50 yards from the working face, without dust suppression, after an average of 6 shots with 9 lbs explosive 222,000 particles in 1 c c. of air (6,313,000,000 in a cu ft.), after an average of 4 shots, with 6 lbs explosive: 105,000 (3,000) With the use of a dust-suppressing device the air was apparently free from dust, but actually, the thermal precipitator showed a concentration of 30,000 (867) At wet drilling apparently no dust was produced, but actually there were over 2,000 (57) very small particles

Methods of working have been changed by replacing, about 1913, the slow hand drilling by machine drilling and the more frequent blasting operations which this change permitted This may be the reason why silicosis had not been generally regarded as an occupational risk until the last few years Collis [31] pointed out the higher mortality for respiratory diseases amongst the Cumberland Lancashire iron ore miners compared with the iron stone (carbonate) workers, in the Registrar-General's statistics for 1910–1912, and Cronin [32] found symptoms and signs of impaired respiratory function in a clinical examination of 100 hematite drillers in 1925 Stewart and Faulds [33] have described 15 fatal cases of pulmonary fibrosis in hematite-miners, and fatal cases have more recently occurred in a haematite mine in the South West of England.

During the six years, 1931–1936, 57 deaths from silicosis were certified, an average of 9 5 per annum

*Prevention* At one time both management and workmen were generally sceptical about the harmful effects of the dust in haematite mines, and accordingly preventive measures

were carried out reluctantly. Since the occurrence of silicosis has been established beyond doubt, strong measures have been taken by the industry to secure suppression of dust in the mines. In one large group of mines a medical officer was appointed to make initial and periodic examinations of the workmen.

The methods relied on include good general ventilation; machine drilling only with the use of axial water feed or other approved method, and hand drilling with the use of water, exclusion of workmen from places after shot-firing until they have been cleared of dust and smoke; the suppression of dust by water where it is liable to be produced in breaking and shovelling rock, and removal of dust from machines used for crushing and dressing.

*Compensation.* The Various Industries (Silicosis) Scheme, 1931 [4] as amended by The Various Industries (Silicosis) Scheme 1935 [34] provides for payment of compensation for disablement or death from silicosis to workmen employed in underground operations in *Hematite Iron Ore Mines.*

### Coal Mining

This is one of the most important industries in the country The situation of the coal-fields and the numbers of wage earners employed at a given date are indicated in Table 3.

While the dusty conditions to which many workers are exposed underground in coal mines has been reflected in the incidence of pulmonary disease in the industry for many years, it is only comparatively recently that the disease now known as silicosis has been identified amongst these workers Silicosis follows especially those processes underground which expose the worker to siliceous dust. These processes are: driving a hard heading or cross measure drift, and ripping or brushing, that is, cutting the roof, floor and sides of a roadway. They involve drilling, shot-firing and loading debris

The following dust-counts are of samples taken by H.H. Watson[35] with the thermal precipitator, in studying the movement of a dust cloud from the point of production along the roadways in a coal mine, while means were not being taken to suppress the dust. The samples were freed from carbonaceous matter before counting. Drilling, usually in sandstone, at 15 yards distance, 10,860 particles in 1 c.c. of air (310,000,000 in 1 cu. ft); at 65 yards, 860 (25); at 160 yards, 630 (18). Shot-firing (with 24 ozs explosive), at 65 yards, 2,000 (57); (with 40 ozs explosive) at 160 yards, 2,300 (65), near the bottom of the upcast shaft, 550 (15).

In 1909 the Royal Commission on Mines[36] referred to the possible danger from dust produced in drilling in siliceous rock in coal-mines, and since 1921 silicosis was found amongst rock-drillers in the coal-mines of Somerset and South Wales, and later in other coal-fields. The distribution of the disease in the different coal-fields has been very unequal, as can be seen from a geographical classification of the certificates issued by the Silicosis Medical Board during the period of four and a half years to the end of 1935 (Table 3). The numbers of certificates issued in respect of fatal cases, if calculated on the number of wage earners at the end of the period is 22.37 for Great Britain, 4.13 for England, and 112.59 for South Wales and Monmouth. No completely satisfactory explanation of this distribution of the disease has yet been reached.

In order to find out the effects on the lungs of coal dust, by itself, the Medical Research Council's Committee on Industrial Pulmonary Disease[37] carried out a clinical and roentgenological examination of 30 coal trimmers employed in loading ships with anthracite coal. The men had been exposed to high concentrations of coal dust for various periods from three years to over forty years, but the Committee did not find any evidence that the inhalation of anthracite or

## TABLE 3

| District | Number of wage earners on Dec 14th 1935 | Number of certificates issued for— | | Suspension |
|---|---|---|---|---|
| | | Death | Total disablement | |
| *England* | | | | |
| Northumberland | 43,865 | | 1 | |
| Durham | 104,244 | | 2 | |
| Cumberland and Westmorland | 4,862 | | | |
| South Yorkshire | 94,427 | 4 | 8 | 10 |
| West Yorkshire | 41,991 | | 3 | 1 |
| Lancashire and Cheshire | 59,607 | 5 | 5 | 5 |
| North Derbyshire | 41,623 | | | |
| Nottingham | 44,832 | 1 | 2 | . |
| South Derbyshire | 3,179 | | | |
| Leicestershire | 9,055 | | | |
| Cannock Chase | 21,366 | 1 | 2 | |
| North Staffordshire | 22,543 | 3 | 16 | 6 |
| South Staffordshire and Worcestershire | 4 460 | | 1 | |
| Shropshire | 2,623 | | 1 | |
| Warwickshire | 16,391 | | | |
| Forest of Dean | 5 075 | | 2 | 2 |
| Bristol | 838 | | | |
| Somerset | 3,410 | 5 | 17 | 7 |
| Kent | 7,303 | 3 | 6 | 4 |
| Total | 531,694 | 22 | 66 | 35 |
| *Wales* | | | | |
| South Wales and Monmouth | 130,552 | 147 | 511 | 198 |
| North Wales | 8 676 | | 1 | |
| Total | 139,228 | 147 | 512 | 198 |
| *Scotland* | | | | |
| Fife and Clackmannan | 21,625 | . | | 1 |
| Lothians (Mid and East) | 12,472 | | 2 | |
| Lanarkshire | 38,830 | | 1 | 3 |
| Ayrshire | 11,311 | | | |
| Total | 84,238 | | 3 | 4 |
| Great Britain | 755,160 | 169 | 581 | 237 |

other coal dust had caused fibrosis of the lungs in this series of cases

Fisher [38] has shown that there is no constant relation between the amount of drilling and the incidence of silicosis The same is true of the practice of stone-dusting It is held by some that a risk of bronchitis is incurred by the men, overheated, travelling out of certain pits by slants through chilling currents of air which reach a velocity of 2500 ft per minute with a temperature drop of 20° to 40° F T. David Jones [37] found that four times as many cases of silicosis were certified

from collieries with slants as from those without slants W.R. Jones[40] stated that the mineral sericite is present in the sandstones of the anthracite coal field, while in those of well-known Scottish collieries, sericite is either absent or rare According to Cooke[41] sericite is constantly present in the atmosphere of the Lancashire coal-mines, but he found that silicosis was extremely rare amongst the coal miners there That "prolonged exposure to the working conditions incident to coal mining is frequently associated with the gradual development of alterations in the radiological appearances of the lungs" is a conclusion reached as a result of a roentgenological study of certain groups of industrially healthy South Wales coal-miners[42] The distinction in the degrees of change found between workers in anthracite coal and in bituminous coal agree with those reported by Russell In the Welsh study the anthracite group showed a degree of "mottling" greater than that found in the semi-bituminous group Harper[43] found no roentgenological difference between cases of silicosis in South Wales and those seen by him in the Belgium coal-fields Lyle Cummins[44] directed attention to the variety of occupations included in the coal-mining industry

In the report of a study made in the Anthracite Region of Pennsylvania by the United States Public Health Service,[45] the results of examination of a total number of 2,711 workers in 3 anthracite coal-mines are described Anthraco-silicosis was found in 23 per cent, and the incidence varied with the period of employment, and the concentration and nature of the dust to which they were exposed Bohme[46] described the results of examination of 3318 rock-drillers working in the Ruhr coal mines for periods from five to twenty-five years Of these, 19 ʇʇ per cent showed changes, due to dust, of varying degrees of severity from slight, 1ʇ 7 per cent to grave, 0 18 per cent

From a study of systematic examinations of 9807 stone

workers in the coal-mines of the Ruhr district Reichmann and Schürmann [47] found the prevalence and degree of severity to be 1048 (1.06 per cent) slight, 89 (0.9 per cent) moderate, and 14 (0.16 per cent) severe. In 493 cases of severe silicosis the duration of employment was, on the average, 18.3 years Death was due to simple silicosis with heart failure in 37 per cent and to an accompanying tuberculosis in 63 per cent

Courtois [48] made a study of pulmonary disease in 136 coal-miners at Charleroi Roentgenological examinations of these workers are classified as negative 33, positive (nodular opacities 30, confluent opacities 61) 91, other conditions (bronchitis, emphysema, and deformities) 12 Post-mortem examination showed that the nodules were typical, simple, silicotic nodules In the massive fibrotic areas the nodular arrangement had disappeared except at the periphery. Leclercq [49] has described the occurrence of silicosis amongst coal-miners in the north of France. Magnin [50] found that the disease produced amongst workers underground in the coal-mines of Central France is silicosis, which occurs in nodular and pseudo tumoral forms. Badham and Taylor [51] found in the coal-miners of New South Wales several different types of pulmonary fibrosis One type was thought to be either a silicatosis due to silicates, or a pure anthracosis due to coal dust.

*Prevention* The Coal Mines Act, 1911, enforces the use of water sprays or jets or other efficient means when drilling in ganister, hard sandstone or highly siliceous rock "the dust from which is liable to give rise to fibroid phthisis" The preventive measures in general use include (1) water, alone or containing substances to increase its wetting power, sprayed over the exposed surfaces in the mines and on dumps of broken material, drilling with hollow or other special drills to which is fed a supply of water, alone or mixed with a foam-producing substance: (2) a device known as a dust-trap, of

which there are large numbers of several types in use, by means of which the dust produced in drilling is drawn by suction into a collecting bag made of dust-proof material (3) respirators, which can be worn where means for suppression or removal of dust are not practicable. (4) efficient ventilation of all parts of the mine workings especially those at which dust is produced by drilling, and fumes and dust are produced by blasting (5) protection from currents of cold air of workmen while being brought out of hot pits on tramways

*Compensation* The Various Industries (Silicosis) Scheme, 1931 [4] as amended by the Order of 22nd October, 1934 [52] provides for the payment of compensation in respect of disablement or death from silicosis for all workmen employed in "*any operation underground in any coal mine*"

## Asbestosis

Asbestosis is described in the Official Memorandum on Silicosis and Asbestosis [53] as a disease of the lungs produced as a result of the inhalation of asbestos dust, usually over a period of several years, and occurs in workers in occupations associated with production of asbestos dust in a fine state of division It is characterised by a special type of fibrosis of the lung tissue which resembles silicosis in its general features of causation and development, but differs from it in some of its clinical and pathological manifestations

Asbestos, which causes the disease, is defined in the Asbestos Industry Regulations 1931 [54] as follows.— "*Asbestos* means any fibrous silicate mineral, and any admixture containing any such mineral whether crude, crushed or opened" This somewhat wide definition is limited in practice, by commercial and industrial usage, to a group of silicates having the common properties of finely fibrous structure and flexibility There are two principal varieties, serpentine asbes

tos or chrysotile, the 'white' asbestos, which is a hydrated silicate of magnesium with a small proportion of iron; and the hornblende minerals, the most important of which are crocidolite, or 'blue' asbestos, and amosite, a brown coloured variety, blue asbestos and amosite consist largely of iron silicates, with a small proportion of magnesium.

Asbestos is imported into Great Britain in increasing quantities, year by year, owing to a remarkably rapid expansion of its uses in industry. The chief sources of supply are Canada, Cape Province, Italy, Rhodesia and Russia. The valuable properties of the fibrous minerals include the facility with which they can be disintegrated into flexible fibres and spun into yarn and woven into cloth, their resistance to heat and electricity confers valuable properties for insulation, and the resistance to acids, of some varieties, favours their use for chemical processes.

The numbers of persons employed in the asbestos manufacturing industries is difficult to compute, but it is probably between 3,000 and 5,000, and they include both sexes The largest section of the industry is engaged in the manufacture of asbestos textiles and the products into which these enter: the processes include the preliminary disintegration of the crude mineral, carding the fibre, spinning, plaiting and weaving the yarn, the making of insulating mattresses of asbestos cloth filled with loose fibre. making brake-linings, fire-proof curtains, clothing, etc. These branches of the industry are largely centred in Lancashire and Yorkshire, and in and near London. Other branches of the industry are connected with building, engineering and shipping, and are spread over the centres where these are carried on. The non-textile products of manufacture include asbestos mill-board; asbestos-cement sheets, tiles and pipes, brake and clutch-linings; packings and jointings, electrodes and switch-board panels. Mixtures of asbestos with magnesia, kieselguhr and other materials are

used as cements or fillings for insulating boilers, engines, pipes and other parts Many persons are employed in handling and applying these mixtures in buildings and ships in course of construction or repair.

The dust given off from asbestos in the processes of manufacture consists of fragments of fibres and small rounded or angular particles The concentration of the dust cloud varies within wide limits, the most important factors being the nature of the process and the variety of asbestos in use In a series of samples of atmospheric dust taken by H H Watson[55] with the thermal precipitator the following concentrations were noted

TABLE 4

| | Process | Asbestos | Position | Particles (in 1 c c) | Fibres (in 1 c c) | Total * |
|---|---|---|---|---|---|---|
| 1 | Carding | White | B L | 235 | 180 | 415 (12) |
| 2 | do | do | G A | 120 | 95 | 215 ( 6) |
| 3 | Doubling | Blue | G A | 105 | 95 | 200 ( 6) |
| 4 | Spinning | do | G A | 115 | 120 | 235 ( 7) |
| 5 | Weaving (cloth) | White | B L | 240 | 85 | 125 ( 9) |
| 6 | do | do | G A | 55 | 40 | 95 ( [illegible]) |
| 7 | Weaving (brake lining) | do | B L | 140 | 70 | 210 ( 6) |
| 8 | do do | do | G A | 130 | 55 | 195 ( 5) |
| 9 | Winding | Blue | B L | 200 | 240 | 440 (11) |
| 10 | Plaiting | White | B I | 120 | 50 | 170 ( 5) |
| 11 | do | do | G A | 120 | 85 | 205 ( 6) |
| 12 | Mattress making | Blue | B L | 405 | 820 | 1 225 (15) |
| 13 | do | Various | G A | 35 | 25 | 60 ( 2) |
| 14 | Bagging | Amosite | G A | 210 | 210 | 470 (12) |
| 15 | Disintegrating | do | G A | 190 | 260 | 450 (13) |
| 16 | Making sections | do | B I | 600 | 1 630 | 2 320 (66) |
| 17 | do | do | G A | 150 | 170 | 120 ( 9) |
| 18 | Pipe making | Blue | B L | 90 | 140 | 290 ( 8) |
| 19 | do | Amosite | B I | 110 | 215 | 325 ( 9) |
| 20 | do | do | G A | 80 | 135 | 215 ( 6) |

B L = breathing level of worker G A = general air of the room
* The figures in parenthesis represent the number of millions of particles in a cubic foot of air

The median length of the *fibres* in 5 counts was between 2 65 μ and 3 5 μ, from 8 per cent to 16 per cent less than 1 μ and 8 per cent to 13 per cent over 10 μ In one sample the thickness of the fibres had a median diameter 0 56 μ, 5 per cent were less than 0 2 μ and 98 per cent less than 2 μ The median sizes of the rounded or angular particles in 5 counts

varied between 0 46 µ and 0 8 µ diameter ; 32 to 54 per cent were less than 0.5 µ and 96 6 to 99 9 per cent. were less than 5 µ

The history of the recognition of asbestosis as a serious industrial disease, and the increase of knowledge regarding it, are described elsewhere, but it may be stated here that the first case recorded in England was that of a card room worker, who died in 1899, the last survivor of 10 men employed in the same room. The case was described by Dr. Montague Murray in 1906 [56] During the same period a similar state of affairs existed in France, when in 1890 a textile asbestos factory was established at Condé-sur-Noireau (Calvados) and 16 workers died of pneumoconiosis between 1890 and 1895 [57]

Merewether [58] examined 363 workers in various processes in the asbestos industry and found in 95, or 26 2 per cent, definite fibrosis due to asbestos dust. Roentgenological signs of a diffuse fibrosis were found in 52 out of 133 persons examined These figures do not represent the average incidence in the industry as a whole, because, for the clinical examinations, workers with longer terms of service were selected, and for the radiological examinations those with some clinical or other suggestive sign were given preference. There was, however, no undue proportion of workers from the more dusty processes. Age, considered apart from duration of exposure, and sex, appeared to have no influence on the onset of fibrosis

During the seven years 1930 to 1936, 78 deaths were certified due to asbestosis in England and Wales 40 of these were women and 38 were men The numbers vary between eight and sixteen in the individual years, but they show no tendency towards diminution in the later years of the period This is due to the delayed development of the disease, which in 42 fatal cases showed an average of twelve years exposure, and the fact that the gravity of the danger from asbestos dust

was not realised until 1924, although, as already mentioned, Montague Murray had recorded a fatal case and referred to others in 1906

*Prevention* The Asbestos Industry Regulations 1931[54] were made following a medical and engineering inquiry into conditions in the industry by Merewether and Price[55] The Regulations apply to all factories and workshops in which any of the following processes are carried on —

(i) breaking crushing, disintegrating, opening and grinding of *asbestos*, and the mixing or sieving of *asbestos* and all processes involving manipulation of *asbestos* incidental thereto,

(ii) all processes in the manufacture of *asbestos textiles* including preparatory and finishing processes,

(iii) the making of insulation slabs or sections, composed wholly or partly of *asbestos* and processes incidental thereto,

(iv) the making or repairing of insulating mattresses, composed wholly or partly of *asbestos*, and processes incidental thereto,

(v) sawing, grinding, turning, abrading and polishing, in the dry state, of articles composed wholly or partly of *asbestos* in the manufacture of such articles,

(vi) the cleaning of any chambers, fixtures and appliances for the collection of *asbestos* dust produced in any of the foregoing processes

The Regulations require that machinery used for processes in the manufacture of asbestos giving rise to dust be so constructed and maintained that dust cannot escape otherwise than by mechanical exhaust draught which prevents the escape of dust into the air of a workroom Mechanical exhaust draught is also required at processes in which asbestos is manipulated

Special requirements apply to the making and repair of

insulating mattresses, one of the most dangerous processes in the industry owing to the difficulty of controlling the finest dust Ventilation of the room must be approved in writing by the Chief Inspector , whilst filling, beating and levelling are being done, persons not actually employed on such work are excluded from the room ; damping of floors, benches and material is prescribed

Cleaning of the cylinders of a carding machine is no longer allowed to be done by hand tools, and persons not engaged *in the work are excluded from the room In practice, suction* apparatus is now used and this is one of the most important preventive measures

A breathing apparatus or approved respirator must be provided for and worn by persons employed in chambers containing loose asbestos, in cleaning dust collecting apparatus, and in filling insulating mattresses. Persons under eighteen years of age must not be employed in any of these processes, or in sack cleaning or blending asbestos by hand.

In all rooms in which the processes are carried on, floors, benches and plant must be kept clean and free from obstructing material or articles not immediately required, and every room must be adequately lighted

Initial and periodical medical examinations may be of great value as a means of preventing or arresting the development of asbestosis. Such examinations are prescribed under the Silicosis and Asbestosis (Medical Arrangements) Scheme, 1931 [26], as amended in 1934 [27], for workmen employed in the processes of breaking, crushing, disintegrating, opening or grinding asbestos, and in mixing or sieving of asbestos or any admixture of asbestos, and in making asbestos *textiles and insulating mattresses The periodic examinations* are carried out by the Silicosis Medical Board at the prescribed intervals of twelve months.

*Compensation.* The Asbestos Industry (Asbestosis)

Scheme, 1931 [59] provides for the payment of compensation by employers of workmen who contract asbestosis due to employment in those processes in the industry in which the disease is liable to occur The processes are as follows — breaking, crushing, disintegrating, opening and grinding of asbestos, mixing or sieving of asbestos or any admixture of asbestos, the manufacture of asbestos textiles, the making of insulation slabs or sections or mattresses composed wholly or partly of asbestos, other manufacturing processes carried on in the same room as any of the foregoing processes, the cleaning of machinery and plant used in the processes, and of chambers, fixtures and appliances for the collection of asbestos dust, the sawing, grinding and turning in the dry state of articles composed wholly or partly of asbestos, except articles composed wholly or partly of woven asbestos impregnated with bitumen or other bond of an adhesive nature

## The Prevention of Silicosis

Silicosis, like every occupational disease, is preventable The long latent period between first exposure and the appearance of symptoms, and the chronic course of the disease tend to obscure the connection between the fatal result and the conditions which brought it about This leads to the argument that the conditions which existed in this or that industry thirty or forty years ago were responsible for most of the silicosis mortality of today That may be true of some industries but conditions do still exist which are capable of producing silicosis and new processes have been introduced without due regard to this danger

The most important measures for preventing silicosis are (1) abandonment of the process, (2) finding a safe substitute for a dangerous material, (3) suppression of dust immediately on its production, (4) removal of dust near its point of origin so that it does not reach breathing level, (5)

exclusion of persons from the room where a dusty process is being carried on, except those actually and necessarily employed in it; (6) protection of individual workmen by respirators or breathing apparatus; (7) medical supervision.

(1) A safe process may be adopted in place of a dangerous one, for example, cleaning metal castings with adherent sand by a wet method instead of by sand- or shot-blasting.

(2) Notable examples of replacing a dangerous substance by a safe substitute are the use of artificial abrasive wheels for sandstone in metal grinding; the use of alumina for placing china-biscuit ware and for polishing and grinding in the pottery industry, metal grit or aluminous abrasive for sand or flint in sand-blasting. These measures have altered the outlook in their respective industries.

(3) Suppression of dust at the point of origin has been achieved to meet the hygienic needs of rapid drilling and other mechanical methods; water alone or combined with foam producers and with suitably modified drills have improved the outlook in mining and quarrying in sandstone, granite and other siliceous rocks and siliceous deposits.

(4) Localised exhaust ventilation by mechanical means is one of the most widely used measures for dust removal. To be effective the plant must be designed to remove the required quota of dust as near as possible to the point of origin and to admit of effective collection and removal. It is a special engineering problem, and its efficiency should be checked at its installation and periodically afterwards by approved scientific instrumental methods of dust estimation Standards of permissible air dustiness are still lacking, but with standardisation of methods these can be evolved, and for the present reasonable arbitrary standards can be maintained. Localised exhaust must usually be combined with some degree of enclosure of machinery which gives rise to dust.

(5) Exclusion from the room of persons not actually en-

gaged in the dusty process is necessary where dust suppression cannot be complete The principle has been incorporated in Regulations and is now specifically included in the Factories Act, 1937, Section 48, as regards periods allowed for meals or rest.

Section 48 of the Act provides that where in any room a process prescribed by Regulations is carried on which gives rise to siliceous dust or asbestos dust, a person shall not be permitted to remain in that room during the intervals allowed to him for meals or rest, and suitable provision must be made to enable such persons to take their meals elsewhere in the factory

Some processes can be carried on mechanically without the presence of any person in the room while the machinery is in motion, for example, crushing and screening of road material. Certain dusty processes required intermittently can be carried out by a minimum number of persons outside ordinary working hours, the persons employed being suitably protected.

(6) Individual workmen can be protected by breathing apparatus providing pure air through a tube or by self-contained oxygen apparatus A certain degree of protection can be secured by efficient respirators, these should, however, have a high filtering efficiency against fine dust, should fit accurately, have a low resistance, and be reasonably comfortable and cheap Protection of this kind cannot be regarded as a substitute for dust suppression or dust removal where such is practicable

(7) Medical supervision of personnel may take the form of selection by initial examination of persons proposed for employment in the dusty process and exclusion of those of deficient respiratory physique or showing any signs of tuberculosis Subsequent periodic medical examinations of persons employed, with roentgenological examinations and the keep

ing of records of the serial examinations; this enables persons showing the earliest evidence of pulmonary changes due to dust, or the onset of tuberculosis, to be suspended from further work in the process This form of supervision can be used as a means of determining the relative dangers of the various processes and the value of measures in use for dust suppression

Section 4 (1) of the Factories Act, 1937[3] requires that effective and suitable provision shall be made for securing and maintaining, by the circulation of fresh air in each workroom, the adequate ventilation of the room, and for rendering harmless, so far as practicable, all fumes, dust and other impurities that may be injurious to health, generated in the course of any process or work carried on in the factory.

Section 47 (1) requires that in every factory in which, in connection with any process carried on, there is given off any dust or fume or other impurity of such a character and to such an extent as to be likely to be injurious or offensive to the persons employed, or any substantial quantity of dust of any kind, all practicable measures shall be taken to protect the persons employed against inhalation of the dust or fume or other impurity, and to prevent its accumulating in any workroom, and in particular, where the nature of the process makes it practicable, exhaust appliances shall be provided and maintained, as near as possible to the point of origin of the dust or fume or other impurity, so as to prevent it entering the air of any workroom

### Compensation for Silicosis and Asbestosis

A beginning was made in providing compensation for these diseases in 1918, when the Workmen's Compensation (Silicosis) Act was passed This Act gave power to the Secretary of State to make schemes for the payment of compensation by the employers of workmen in any specified industry or

process or group of industries or processes involving exposure to silica dust, who were certified to have suffered death or total disablement from silicosis or silicosis accompanied by tuberculosis, or who, though not totally disabled, were found on medical examination to be suffering from the disease to such a degree as to make it dangerous to continue work in the industry and were for that reason suspended from employment.

The Act required that the scales of compensation fixed by any scheme, in the case of death or disablement from silicosis, should be those prescribed by the Workmen's Compensation Act, 1906, in all other cases such as may be prescribed in the Scheme. The Act further provided for the inclusion in any Scheme of 3 principles :—

(1) The establishment of a general compensation fund (administered either through a mutual trade insurance company or society of employers) to which all the employers in the industries should be required to subscribe and out of which all claims for compensation and all expenses arising under the Scheme should be paid. The burden of compensation would thus be borne by the industry as a whole and no question of apportioning the liability among different employers would arise ; further, there would be no tendency to dismiss particular workmen who showed symptoms which might indicate the onset of the disease.

(2) The settlement of claims, and other matters arising under the Scheme, by Committees representative of both employers and workmen with an independent chairman.

(3) The appointment of medical officers with special knowledge of respiratory diseases, who would carry out periodic medical examinations and have power to suspend any workmen from further employment in the industries ; and the appointment of Medical Advisory Committees or Medical Referees for the purpose of deciding difficult cases referred

to them by the medical officers on whose certificate only compensation could be paid.

By the inclusion of these three principles the special difficulties in the way of providing compensation for silicosis were met. These had been recognised and pointed out by the Committee on Compensation for Industrial Diseases in 1906. They found there would be difficulty in apportioning liability amongst employers for a slowly developing disease occurring in workmen who had moved from one employment to another; difficulty in diagnosis of silicosis from respiratory affections generally prevalent; and a possible tendency amongst certain employers to dismiss workmen showing any symptoms, in order to avoid liability.

The first Scheme under the Act of 1918 was made for the Refractories Industries and came into force on February 1, 1919. This Scheme embodied the 3 principles outlined above. The medical officers appointed under the Scheme were generally the Tuberculosis Officers of the Local Authority administering sanatorium benefit under the National Health Insurance Act. The duties of the medical officers laid down in the Scheme were (1) to make periodic medical examinations of all workmen in the industries, except those employed over twenty years, at specified intervals of one year in the more dangerous processes, and two years in the others; (2) to examine newly engaged workers within three months of commencing work in the industry; (3) to examine on the warrant of a Joint Committee any workman claiming to be suffering from the disease; (4) to examine at specified intervals workmen in receipt of payments for partial disablement. If at either periodical or initial examination a work-

sary. If still in doubt about the diagnosis he could refer the case to the Medical Advisory Committee or Medical Referee, after which he issued a certificate in accordance with their decision. The medical officers forwarded their certificates to the Joint Committee, whose duty it was to determine the awards of compensation.

The Refractories Industries Scheme, 1918, was a new and interesting experiment in Workmen's Compensation, and it appeared to work smoothly and to give rise to no serious dispute.

A Departmental Committee was appointed by the Home Secretary in 1923[60], to inquire into the working of the Scheme and to advise on any proposals for applying the Act to other industries. This Committee found certain defects for which they suggested improvements, and they considered that the Scheme, if amended in accordance with their recommendations, would form a basis on which to frame similar schemes for other industries in which a risk of silicosis occurs. In particular, they referred to the absence of power to suspend persons suffering from tuberculosis without silicosis; the lack of uniformity in the medical examinations; failure to eliminate workmen showing early signs of disease; and the difficult financial position of the Fund.

In their Report the Committee sought to remedy the defects in the medical provisions by recommending that a Medical Board be established consisting of two whole-time Medical Officers who should be responsible for all medical examinations; facilities for roentgenologic examinations should be increased and greater use made of this form of examination; the responsibility of suspending a workman on account of silicosis should not be entirely on one medical officer; power should be given to medical officers to suspend workmen found to be suffering from tuberculosis of the lungs (though no compensation was payable); entrants to the

industry should reach a prescribed standard of respiratory physique The recommendations of the Committee were adopted and included in an amending Act passed in August, 1924, and later in Section 47 of the Workmen's Compensation Act, 1925.

In 1929 The Sandstone Industry (Silicosis) Scheme was made on the lines of the Scheme for the Refractories Industries *The same principles were adopted for the provision and* maintenance of the Fund from which compensation and expenses were paid; Joint Committees settled claims and certificates were given by members of the Medical Board who, with an increased staff, also made initial and periodical medical examinations.

These two Schemes, for the Refractories Industries and the Sandstone Industry, are the only Schemes of that type.

In 1927, the Metal Grinding Industries (Silicosis) Scheme was made to apply to workmen employed in the grinding of metals, incidental work in the same room; and the racing of grindstones In 1928 The Various Industries (Silicosis) Scheme was made to apply to workmen in a large number of industries and processes, not covered by the existing Schemes, in which the risk of contracting silicosis was recognised. The machinery under these Schemes, which at first were limited to cases of death and total disablement, was that applicable to diseases scheduled under the Workmen's Compensation Act, namely, examination and certification by *the Certifying Factory Surgeon* for the district in which the workman was employed, subject to a right of appeal to a Medical Referee Except for certain occupations in the pottery industry, no periodical *medical examinations were made.*

A Departmental Committee was appointed by the Home Secretary in December 1928, to advise as to the medical arrangements which could be made for the diagnosis of silicosis in cases of claims arising under the Workmen's Com-

pensation Act [61], and for carrying out any periodical or other medical examinations of workers which may be prescribed for any industry or process involving risk of silicosis. This Committee recommended that the examinations and certifications in the industries covered by all the Schemes be co-ordinated and a Medical Board set up for the purpose, of which the Medical Board already appointed under the Refractories Industries and Sandstone Industry Schemes would form the nucleus. They considered that where compensation was claimed in fatal cases, a certificate as to the cause of death should only be given after a post-mortem examination, except where the deceased workman was in receipt of weekly payments under the Scheme, and then only if the Medical Board were satisfied as to the cause of death without a post mortem examination. Power to carry out the recommendations of the Committee was obtained in the Workmen's Compensation (Silicosis and Asbestosis) Act, 1930, which extends Section 47 of the Workmen's Compensation Act, 1925 A Medical Arrangements Scheme followed in 1931 [26], and the existing Schemes were amended to give effect to its requirements as to the Medical Board, Medical Examinations and Certificates, and Medical expenses and fees The fees payable for examinations and certificates by the Medical Board are prescribed by the Silicosis and Asbestosis (Medical Fees) Regulations [62].

The Medical Board now consists of 9 members . the Chief Medical Officer with a head office and laboratory at Sheffield, and 4 panels each of 2 medical officers, situated at centres convenient to the principal industries to which the Acts apply These are Sheffield, the centre of the Refractories and Metal Grinding industries, Manchester, dealing principally with the Asbestos industry in Lancashire, and the Sandstone industry of the North of England and Scotland, Stoke-on Trent, the centre of the Pottery industry, and Cardiff, a con

venient centre for the South Wales coalfield from which most of the cases of silicosis occurring in the coal-mining industry arise. Besides the whole-time members of the Medical Board, a few part-time members have been appointed to act, especially in fatal cases, in areas at a distance from the centres of the Board. Certificates are issued on the authority of not less than 2 members of the Board, and are conclusive.

The 1930 Act also extended the power of making Compensation Schemes to the disease of asbestosis, and a Scheme on the lines of The Various Industries (Silicosis) Scheme was made for the Asbestos Industry and came into force on 1st June, 1931. The provisions of the Silicosis and Asbestosis (Medical Arrangements) Scheme apply also to examinations and certificates required for the purposes of this Scheme, and the examinations are carried out by the same Medical Board.

## TABLE 5

### Number and Results of Examinations under the Silicosis and Asbestosis (Medical Arrangements) Scheme, 1931, during the Year 1935.

#### (A) Initial and Periodic Examinations

| Industry | Initial Examinations | | Periodic Examinations. | | | |
|---|---|---|---|---|---|---|
| | | | | | Number suspended | |
| | Examined | Suspended | Clinical examinations | Radiological examinations | On account of the disease * | On account of tuberculosis |
| Refractories industries | 582 | 33 | 605 | 117 | 5 | 1 |
| Sandstone industry | 645 | 44 | 2,028 | 385 | 22 | – |
| Pottery industry | 473 | 31 | 1,394 | 215 | 15 | 1 |
| Asbestos industry | 438 | 24 | 1,099 | 153 | 32 | – |
| Total for 1935 | 2138* | 132 | 5,126 | 867 | 74 | 2 |
| Total for 1934 | 1639 | 100 | 6,451 | 1 058 | 63 | 12 |

* 540 of these examinations were made by local tuberculosis officers, 23 being referred to the Medical Board for suspension

## (B) Examinations in pursuance of applications for Death and Disablement Certificates

| Industry | Disablement | | Death | |
|---|---|---|---|---|
| | Workmen examined | Certified to be wholly or partially disabled by the disease * | Applications dealt with | Certified to be caused by the disease * |
| Refractories industries | 14 | 7 | 10 | 7 |
| Sandstone industry | 53 | 41 | 51 | 43 |
| Pottery industry | 128 | 66 | 39 | 32 |
| Asbestos industry | 9 | 4 | 2 | 1 |
| Coal mining industry | 492 | 228 | 69 | 51 |
| Masons & stone dressers | 73 | 49 | 35 | 23 |
| Metal grinding industries | 16 | 13 | 10 | 8 |
| Other industries | 51 | 31 | 12 | 10 |
| Total for 1935 | 836 | 439 | 228 | 175 |
| Total for 1934 | 690 | 417 | 203 | 143 |

* The disease means silicosis or asbestosis or either of these diseases accompanied by tuberculosis

Home Office Workmen's Compensation Statistics of Compensation etc, in Great Britain during the year 1935 London H M Stationery Office, 1937

The scope and amount of work done by the Silicosis Medical Board can be gathered from the preceding Tables, which give details of the examinations in the year 1935, with comparative figures for 1934

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6 Statutory Rules & Orders, 1925, No 1089 H M Stationery Office, London

7 Report on the Grinding of Metals & Cleaning of Castings,

venient centre for the South Wales coalfield from which most of the cases of silicosis occurring in the coal-mining industry arise Besides the whole-time members of the Medical Board, a few part-time members have been appointed to act, especially in fatal cases, in areas at a distance from the centres of the Board Certificates are issued on the authority of not less than 2 members of the Board, and are conclusive.

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| | | | | | On account of the disease + | On account of tuberculosis |
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| Sandstone industry | 645 | 44 | 2,028 | 385 | 22 | – |
| Pottery industry | 473 | 31 | 1,394 | 215 | 15 | 1 |
| Asbestos industry | 488 | 24 | 1,099 | 150 | 32 | – |
| Total for 1935 | 2188× | 132 | 5,126 | 867 | 74 | 2 |
| Total for 1934 | 1639 | 100 | 6,451 | 1,058 | 63 | 12 |

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## (B) Examinations in pursuance of applications for Death and Disablement Certificates

| Industry | Disablement | | Death | |
|---|---|---|---|---|
| | Workmen examined. | Certified to be wholly or partially disabled by the disease * | Applications dealt with | Certified to be caused by the disease * |
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| Sandstone industry | 53 | 41 | 51 | 43 |
| Pottery industry | 129 | 66 | 39 | 32 |
| Asbestos industry | 9 | 4 | 2 | 1 |
| Coal mining industry | 472 | 228 | 69 | 51 |
| Masons & stone dressers | 73 | 49 | 35 | 23 |
| Metal grinding industries | 36 | 13 | 10 | 8 |
| Other industries | 51 | 31 | 12 | 10 |
| Total for 1935 | 836 | 439 | 228 | 175 |
| Total for 1934 | 690 | 417 | 203 | 149 |

* The disease means silicosis or asbestosis or either of these diseases accompanied by tuberculosis

Home Office Workmen's Compensation Statistics of Compensation etc, in Great Britain during the year 1935 London H M Stationery Office, 1937

The scope and amount of work done by the Silicosis Medical Board can be gathered from the preceding Tables, which give details of the examinations in the year 1935, with comparative figures for 1934.

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## VII. PUBLIC HEALTH AND ECONOMIC ASPECTS
## *UNITED STATES*

A. J. LANZA, M.D.
ASSISTANT MEDICAL DIRECTOR
METROPOLITAN LIFE INSURANCE COMPANY

### INTRODUCTION

THE occupational diseases and the health of industrial workers present one of the important phases of public health. Of the occupational diseases, none is more truly occupational than the pulmonary diseases due to the inhalation of industrial dusts Lead, carbon monoxide, arsenic, benzol and many other toxic substances associated with industrial processes are also found in circumstances that have no connection with industry The same cannot be said for the pneumoconioses They stand as the most perfect type of occupational diseases Recognized for centuries, only in comparatively recent times have their wide-spread effects been disclosed and their importance to the public health appreciated More lately still, the inclusion of these disorders in schemes of compensation for occupational disease has emphasized their economic importance and the necessity for thorough and adequate methods for their control.

### SILICOSIS AND COMPENSATION

Silicosis and asbestosis burst upon the amazed consciousness of American industry during the period 1929–1930 Previously, the terms "silicosis", "asbestosis", "pneumoconiosis", were practically unknown to industrialists except that in the hard rock mining industry, silicosis, under its various colloquial designations, was well recognized Knowledge of these

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### Introduction

The occupational diseases and the health of industrial workers present one of the important phases of public health. Of the occupational diseases, none is more truly occupational than the pulmonary diseases due to the inhalation of industrial dusts. Lead, carbon monoxide, arsenic, benzol and many other toxic substances associated with industrial processes are also found in circumstances that have no connection with industry. The same cannot be said for the pneumoconioses. They stand as the most perfect type of occupational diseases. Recognized for centuries, only in comparatively recent times have their wide-spread effects been disclosed and their importance to the public health appreciated. More lately still, the inclusion of these disorders in schemes of compensation for occupational disease has emphasized their economic importance and the necessity for thorough and adequate methods for their control.

### Silicosis and Compensation

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conditions was limited to the small number of those whose investigations of pulmonary dust diseases had made them conversant with the subject.

Arising out of the period of economic depression, the situation with respect to silicosis and asbestosis became manifest as a medico-legal phenomenon of a scope and intensity that was at once preposterous and almost unbelievable. Damage suits, under the common law, were instituted against employers by employees, alleging pulmonary dust diseases, in industrial centers all over the United States, to an amount in excess of 100,000,000 dollars. To understand this situation, both in its economic and public health aspects, it is necessary to review not only the history of silicosis investigations in the United States but also the status of compensation for occupational diseases in the various States.

The reader is referred to the first chapter of this book. Briefly, governmental agencies began studies of silicosis in the mining industry in 1914 and these were carried on until they were interrupted by the entry of the United States into the World War. These studies had for their purpose the detection of silicosis, the extent and severity of the disease in the areas studied and the establishment of methods of control The question of compensation did not arise, which must seem strange to the non-American student of the subject.

In almost every State of the Union, there is a State Department of Labor and Industry These State Departments are charged with the administration of the laws pertaining to factories, mines, and workshops Previous to 1930 only 3 or 4 Labor Departments included in their activities a division of industrial hygiene and these few were not established on a basis that made possible original studies or research They were mostly concerned with routine inspections and also endeavored to enforce laws and regulations pertaining to

health, whose vague wording often made them almost non-enforceable

The State Departments of Health, although their functions might be thought to embrace public health in industry, uniformly ignored the subject altogether, with the exception of Ohio and Connecticut, where occupational diseases were made reportable. Following the widespread national attention focussed upon occupational diseases by the silicosis furor, this situation has entirely changed and at present (1937) State Divisions of Industrial Hygiene are, with a few exceptions, located in the State Departments of Health

The Federal Government embraces several agencies that concern themselves with occupational diseases and industrial hygiene. In the United States Department of Labor, the Bureau of Vital Statistics has been actively engaged in the field of investigation. In the United States Public Health Service, the Office of Industrial Hygiene and Sanitation has likewise been engaged in industrial studies and at the request of and in co-operation with the United States Bureau of Mines investigated dust diseases These governmental departments have jurisdiction in industrial establishments of a governmental nature only, except that by virtue of recent legislation, the United States Department of Labor may supervise conditions under which work is done under a contract with the Federal Government The Government officers enter a mine or a factory only with the permission of the owners and usually at their request The Federal officials have no power to enforce existing State laws nor to initiate new laws or regulations Reports of silicosis investigations, when published by the department responsible, might and did contain suggested methods for the prevention and control of this disease but they were addressed not to the State authorities nor to the industrialist but to the general public as contributions to the

existing knowledge. Both State officials and industrialists might profit by this new knowledge if they so wished. Usually they did so and a great many improvements were instituted but these were largely confined to those industries that had been studied at first hand. In fact, during the period 1914-1929, silicosis was considered a serious hazard only in certain phases of the mineral industry, with the exception of a few specific occupations, such as sandblasting Asbestosis was practically unknown.

To summarize, the investigations of silicosis during the period 1914-1930, and of both silicosis and asbestosis for some time subsequently, were scientific (embracing both medical and engineering aspects), informative and non-legislative, with the exception of the study of silicosis among rock drillers made in New York City in 1928 under the auspices of the New York Tuberculosis and Health Association[1] In this instance, definite recommendations regarding workmen's compensation were made

Since 1912, in Massachusetts, occupational diseases have been compensated by court interpretation of the wording of the accident compensation law. In 1914, no other State had made provision for compensation for occupational diseases. By 1930, however, 10 States and 3 Territories had enacted laws making occupational diseases compensable.[2] In addition, there were 3 Federal laws, affecting employees of or under the jurisdiction of the United States Government. At present (November, 1937), 21 States have some form of occupational disease compensation and of these, 16 include silicosis and other dust diseases. When the time came to enact legislation bringing occupational diseases within the scope of the compensation acts, some of the States followed British precedent and enacted a schedule of the diseases which were thus made compensable. In the other States, occupational diseases were covered under a general description such as 'diseases arising

out of and in the course of occupation,' without further designation — the so called 'all inclusive' or 'blanket law' The Federal Government followed the latter method in providing compensation for its employees

Between 1914 and 1930, the total amount of compensation paid for occupational diseases was very small, averaging not more than a minor fraction of the accident compensation cost in the various States Interestingly enough, up to 1935, not one State operating under a schedule included silicosis, asbestosis, or pneumoconiosis in the list of compensable diseases, a situation difficult to understand, considering that silicosis is *the most truly occupational of diseases*, that reports of studies of silicosis in the United States had been forthcoming for twenty years and that there was available a vast amount of literature embodying the experience of England, South Africa, Australia, and Canada In the States having 'all inclusive' coverage, the number of awards for silicosis was negligible

The explanation of this curious state of affairs is, probably, that silicosis was considered as distinctly a hazard of mining In those mining States, where hard rock mining was a major industry, there was no compensation for occupational diseases nor was there any pronounced demand for such compensation 'Eating dust' was looked upon by the hard rock miner as a natural danger of his trade Mining companies usually pensioned older employees disabled with silicosis or saw that they were cared for in one way or another Also, following the governmental reports, the mining companies concerned started to put their mines in order, spending large sums of money on ventilation and dust control

Then, with dramatic suddenness, came the era of the silicosis damage suits. Lawyers who specialized in this type of law practice instituted suits for damages under the common law on behalf of employees of various corporations for injury to the lungs due to the inhalation of dust They were aided not

only by the general and increasing unemployment but by the almost total ignorance of industry — aside from mining — concerning pulmonary dust diseases and by the absence of silicosis in the compensation schedules which enabled them to bring suits for amounts far in excess of the limits of compensation An interesting exception occurred in one or two States — Ohio, for instance — where the absence of silicosis from the schedule was held to bar suits or claims for this disease either under compensation law or common law. In those States where there were no occupational disease laws, claimants were at liberty to sue under the common law.

Among the first claims for damages were those for asbestosis, a hitherto unheard of disease in the United States [3] Damage suits multiplied by hundreds, starting on the Atlantic seaboard and extending to the industrial States of the middle West and far West The terms 'pneumoconiosis,' 'silicosis,' 'asbestosis' were strange and unfamiliar to judges, juries, and industrialists and were enveloped in medical and pathological technicalities Consequently, the claimant's witnesses and medical experts, as far as their prestige with juries was concerned, carried as much weight as did those of the defendants So great was the confusion and lack of knowledge on the part of courts, juries, and physicians that awards were made for silicosis to men who had never been exposed to the dust of free silica Many of those seeking damages undoubtedly had silicosis — many did not Damage suits were filed against cement companies and against mining companies whose operations were in limestone formations where there could be no possibility of a silicosis hazard

All of this might be dismissed as a historical and medico legal curiosity except that it greatly modified existing compensation laws in the United States and stimulated the passage of new laws Commissions were appointed by several of the States to inquire into and recommend legislation for compen

sation of occupational diseases and especially silicosis It was generally felt that it was not sufficient merely to add silicosis to an existing schedule or include it when a new schedule was planned but it was essential to make special provision for regulating and limiting the benefits to be paid for silicosis. Many industries had acquired through the years considerable numbers of employees who had, or might be deemed to have, silicosis, industrialists, insurance companies, and the State funds feared that simply to make silicosis compensable was to invite a deluge of claims that would exhaust existing funds and make insurance rates prohibitively high In most of the enacted laws, a fixed period of residence within the State was stipulated to prevent silicotics from migrating from a State where there was no compensation to one where there was

The problem of compensation for a chronic illness as contrasted with an acute one presented difficulties, inasmuch as it did not seem that the insurance principle, on which all compensation in the United States is based, could be successfully applied to disability or to diseases already existent and whose extent was problematical With business conditions as they were, the industries did not feel that they should be compelled to establish special silicosis benefit funds to provide for accumulated cases which had occurred under conditions where there was no statutory requirement imposing an obligation upon the employer which he had failed to meet Moreover, it was often evident that silicotics had not contracted their disease in the place of present employment but in a previous one and, perhaps, in another State The attitude of the average industrial firm was that, being shown that they had a silicosis hazard, they were willing and anxious to clean up and endeavor to control dust and to pay compensation to those who might subsequently contract silicosis but that they should not be required to pay for disability from a disease of whose existence they were previously unaware The various commissions

and legislative committees endeavored to iron out these complexities as best they could, having in mind their own local conditions

Some years previously, the Province of Ontario had assumed the attitude that, tuberculosis being a disease common to the human race generally, when silicotics contracted tuberculosis, their compensation benefits for disability should be diminished and such a provision was enacted into law.[4] A similar economic point of view, regardless of its logic or justice, influenced those engaged in drafting legislation in the various States, so that when silicosis legislation was finally enacted, in some of the States the individual disabled by silicosis received less award than one suffering from any other compensable occupational disease. Furthermore, several of the State Committees took the position that uncomplicated silicosis, as observed in American industries, was not disabling and that disability did not occur until infection (tuberculous) had supervened and then the disability was considered total Hence, in some States, partial disability due to silicosis is not recognized.

The New York law,[5] as in some other States, provides that compensation shall not be paid for partial disability due to silicosis or other dust diseases. For permanent disability or death, the awards are fixed according to a sliding scale as follows: For a total disability or a death claim, presented within the first month in which the Act becomes effective, not exceeding $500, for every subsequent month thereafter, $50 is added, but in no event shall the compensation exceed the aggregate of $3000. The maximum for total disability from other occupational diseases or from accidents may be considerably more, depending upon the age of the deceased and upon the number of dependents.

It was hoped that the sliding scale would have a twofold effect It would safeguard the continuity of employment by

spreading the economic burden which was expected to result from these laws and it would also tend to induce the workman with an early silicosis to keep on working. In addition, it would stimulate the employer to vigorously attack his dust problem with the hope of averting total disability claims.

In Pennsylvania, the occupational disease law [6] provides that for diseases which develop to a point of disablement only after an exposure of five years (to be determined by the Workmen's Compensation Board), compensation costs for disability or death, arising during the period of ten years succeeding the effective date of the Act, shall be paid jointly by the Commonwealth and the employer as follows: In the first year in which the Act becomes effective, one tenth by the employer and the remainder out of funds controlled by the Commonwealth. In each succeeding year, the employer pays one tenth more, the Commonwealth one tenth less, to the expiration of the ten year period. Partial disability for silicosis, anthraco-silicosis, and asbestosis is not recognized and the total liability of the employer for these diseases is limited to $3600.

In North Carolina, the recently enacted compensation law [7] states that in silicosis or asbestosis, compensation shall be paid as provided in the Act but if either is complicated by tuberculosis, the rate of payment may be reduced one-sixth.

While some of the foregoing might seem to lie in that realm of reasoning so difficult of comprehension to the non-legal mind, the underlying motive was not a desire to deprive working men of their just dues but a feeling that it was necessary to place a drastic check on the expected avalanche of silicotic claims. Undoubtedly, the common law damage suits confused and terrified industrialists and insurance officials. As a matter of fact, many of these suits were never brought to trial, many were compromised by settlements out of court, but those that did come to trial were won by claimants in sufficient number

to inspire dread among all exposed to this type of legal action. If these apprehensions prove to have been unwarranted it is probable that in the next few years, Silicosis Acts may be modified to make their awards more in line with the general trend of compensation.

The North Carolina Act also stated that if an employee is affected by silicosis or asbestosis, he shall be taken out of employment and paid partial or total disability until he can obtain other occupation. The State will train him for other occupation at State expense. If later he returns to a job with a silicosis hazard, he forfeits compensation benefits.

The experience of Wisconsin is very instructive and for a description of the complexities of silicosis compensation in one State, the reader is referred to the informative article by Kossons and Fried,[8] from which the following figures were obtained. Wisconsin is an important industrial State with mines, quarries, foundries, and enamel ware industries, besides many others. During the seventeen year period — 1920–1936 inclusive — 893 claims for disability from silicosis were brought before the Industrial Commission. Of these, 469 claims were allowed (one third of them were death claims) and 55 per cent of these were filed during 1933–1934. A comparison of costs for the fifteen years — 1921–1935 — shows that for the entire period, the cost of silicosis compensation was 2.23 per cent of the total cost, including and in spite of the peak years of 1933–1934, when it was 9.40 per cent and 8.80 per cent respectively.

## Extent and Nature of Hazard

It is not possible to estimate accurately the number of persons exposed to the harmful effects of silica dust. Comprehensive statistical information, based upon accurate death records, is lacking. Silicosis is not included in the list of reportable diseases. Many of the deaths due to silicosis with

erculosis have been recorded simply as tuberculosis Neither hospital records nor death records customarily give a workman's industrial history — indeed, it is unusual for even present or final occupation to be stated with sufficient clarity to be of value to the investigator. Morbidity records are of less avail than mortality records, and physical examinations of new employees in industrial establishments, while fairly common, have rarely been of such a nature as to reveal early pulmonary disease Even when industrial conditions are good, there is considerable movement of labor from place to place and from State to State When times are bad, this migration is increased Unemployment then drives men not only from place to place but into new occupations

Lanza and Vane [9] estimated that there were about 500,000 industrial workers employed in occupations exposed to silica dust It is more than likely that this estimate errs considerably on the conservative side This estimate does not take into consideration the thousands of men who have been employed where there was a silica hazard and who are now scattered throughout industry.

The menace in silicosis has always been tubercle infection The evidence [10] here is overwhelming and has been stressed by investigators throughout the world In 1927, Watkins Pitchford [11] stated "In ascertaining, from our records, what the liability of the simply silicotic miner to become tuberculous has been in the past, account has of course been taken of the instances in which the change has been detected during life, in addition to this, however, it has been assumed that, in those persons suffering from simple silicosis who have died of their disease without having been again examined, death has been the result of this change This is an assumption which is warranted in practically every instance Relying on this assumption, and also bearing in mind that, as already stated, the liability to tuberculous change has appeared less marked

in each succeeding annual batch of cases, one finds that the combined results of *seven years' observations show that, by* the end of the seventh year, a little more than half of the cases of simple silicosis have either developed overt tuberculous changes or have died as a result of those changes."

Collis [12] also bears witness: "Silica dust alone possesses the further characteristic that it increases the liability to succumb also to pulmonary tuberculosis."

Kettle [13] states the case very clearly: "The silicotic lung appears to be a more favorable medium for the growth of the tubercle bacillus than the normal lung, not because it is fibrotic, *nor because its lymphatic drainage is interfered with,* but because it contains silica."

Much research and study have been applied to the question of infection in silicosis but in spite of the fact that the nature of the relationship between silica and the tubercle bacillus is still to be determined, the reality of this relationship is affirmed by recent investigators. Irvine [14] and his associates analyzed 543 deaths in 1923, cases originally certified as having simple silicosis Approximately 75 per cent of these deaths were due to tuberculosis They note that this inherent predisposition *to tuberculosis is as marked in very early* (antеprimary) as in later stages (primary).

Simson and Strachan [15] state: 'The importance of the infective factor in many and ultimately in the late stages of most cases of silicosis has always been recognized in South Africa, as elsewhere.' They comment further that many cases are infective from the outset, in others there may be a reactivation and local extension of pre-existing dormant foci of infection or there may be a new infection from outside.

The experience in the United States is similar. Meriwether [16] states for the Picher, Oklahoma District 'The production rate of tuberculosis (exclusive of old cases that progressed) was 0 75 per cent or 750 per 100,000 population,

which, for 1929, is certainly higher than that of the general population of the country."

The following is quoted from the United States Public Health Service report on Anthraco-Silicosis among Hard Coal Miners:[17] "Several surveys have shown that tuberculosis of the lungs occurs among 1 to 2½ per cent of the general adult white male population of the country. In a study of tuberculosis in Framingham, Mass., it was found that about 1 per cent were suffering from the disease in an active form, and another 1 per cent were classified as having arrested tuberculosis.[18] Physical examination of 100,921 adult white males made by the Life Extension Institute indicated a prevalence rate of about 1½ per cent when suspected cases were included.[19] A somewhat higher percentage, namely, 2½ per cent, was found by the Public Health Service from the examination of 10,000 male industrial workers.[20]

"Among the anthracite workers examined, the clinical tuberculosis rate was below normal in the younger adult ages, but at ages 35 to 44 clinical pulmonary tuberculosis was diagnosed in about 5 per cent of the hard coal mining employees, at ages 45 to 54 in 10 per cent, and at ages 55 to 64 in 20 per cent. No such rise with age occurred in any general population group for which comparable data are available.

"The prevalence of tuberculosis was greatest among the rock workers. The next to the highest rate occurred among anthracite workers who had changed more than five years previously from very dusty to relatively nondusty occupations in the industry. The third highest rate was exhibited among persons who had had appreciable exposure to harmful dusts in other industries. Among the regular miners working at the face the rate was definitely higher than in the control group which showed a prevalence rate of less than 1 per cent.

"When the term of service exceeded twenty years, more than two or three of which involved exposure to heavy concen-

trations of rock dust, about 37 per cent of such employees (classified as rock workers) showed evidence of pulmonary tuberculosis Service of twenty-five to thirty-four years was associated with a tuberculosis rate of 8 per cent among nonrock workers employed in the haulageways, of 14 per cent among the regular miners, but with a rate under 2 per cent among men exposed to less than 5,000,000 dust particles per cubic foot of air."

As has been pointed out on numerous occasions, the severity of an individual case of silicosis depends upon the extent of dosage of silica ; that is to say, upon the duration of exposure together with the intensity of silica dust concentrations in the air The early investigators of silicosis in the mining industry in the United States were confronted by working conditions of the utmost hazard — that is, a maximum amount of silica dust in the air with no provisions for its control nor for the protection of the men exposed Hazards of this severity have happily disappeared except, perhaps, in isolated instances, certainly such conditions are no longer industry-wide.

Some recent investigations reveal a degree of exposure which is manifested as a disabling illness after middle age has set in — characteristic of certain phases of the foundry industry [21] Again other studies have shown the existence of silica hazards sufficient to produce characteristic roentgen ray appearance but without disability that can be directly charged against pulmonary fibrosis. It seems fairly certain that the industrial hygienist of the future will not see the typical cases of far advanced silicosis, presenting roentgenograms with "snow storm" appearance and areas of massive fibrosis described by early investigators both in this country and abroad

On the other hand, the tendency to minimize the silica dust hazard which has been manifested on recent occasions is not justifiable. If there is one thing that stands out with con-

spicuous regularity in the vital statistics of this and other countries, it is the high incidence of tuberculosis and other respiratory diseases in the silica dust trades[9, 22] We know that where silica has been inhaled in sufficient quantity to produce its characteristic effect, a definite condition of susceptibility to tuberculosis has been established We should hesitate to infer, therefore, that exposure to silica dust to an extent insufficient to produce characteristic nodulation may not carry with it implications in terms of tuberculosis and other respiratory diseases From a public health standpoint and for the promotion of healthful conditions of work, there is demanded constant attention to dust hazards in industry with the utilization of every practical method for the prevention of atmospheric pollution

## ASBESTOSIS

Asbestosis was first described in the United States by Pancoast, Miller, Smyth and Landis in 1918, but its importance was not realized until it was introduced into the damage suit situation which led to the first comprehensive study of this disease in the United States[3] Recent years have seen an immense increase in the use of asbestos, both for brake linings of automobiles and for heat resisting and insulating materials. With relation to public health, asbestosis is not nearly as serious a problem as silicosis The number of persons in the industries using asbestos is very much less than for silica and in the United States amounts to about 12,000 factory workers, of whom approximately 25 per cent are women Not all of these are exposed to dusty processes

Most of the asbestos used in the United States is imported from Canada It has been suggested that the various types of asbestos differ in their ability to cause pulmonary disease, but so far no proof of this is forthcoming

Much of the industrial exposure arises from textile proc

esses in which dust control measures can be readily instituted. As soon as the hazard was realized, industrial firms fabricating asbestos took energetic steps to control the dust so that it is probable that cases of asbestosis will become uncommon. A recent report from the United States Public Health Service details dust control methods instituted in an asbestos textile plant which illustrates in a striking manner what can be done to alleviate dust [23]

Asbestosis has not appeared to be as serious a disease in the United States as it has in England, judging from the various reports from the latter country. Asbestos "mattress making," mentioned in the British reports as a serious hazard, is a process not found in this country The role of infection in asbestosis is not as clearly defined as in silicosis and the clinical evidence available indicates that tuberculosis does not have the intimate relationship with asbestosis that is so conspicuous in silicosis [24 25]

## Effects of Silicosis upon the Families of Silicotics

From the earliest times, when silicosis was first described as a disease of miners, the fact that the wife did not contract tuberculosis from the husband was mentioned and the many recent investigations have not upset this general conclusion. Hoffman [26] stated in 1922, "The frequency of pulmonary tuberculosis among immediate relatives of granite cutters, but particularly wives, daughters, and mothers, is much less than, as a mere matter of probability, would be assumed to be the case." This was borne out by the later studies of McFarland [27] and Russell [28]

Cummings of the Saranac Laboratory, who investigated this subject in connection with the Picher Clinic, commented on the fact that, despite the wretched living conditions, and the lack of cleanliness and sanitation in many homes of advanced silicotics whose sputum was loaded with tubercle bacilli, no

adult type tuberculosis was found among exposed children up to the age of sixteen.

Nevertheless, these various testimonies do not exclude the necessity of treating tuberculo-silicosis as a communicable disease and of making follow-up investigations of members of families of advanced or disabled silicotics, to determine the presence of tubercle infection or the desirability of segregating contacts from the diseased member of the family. Sound public health procedure should be the rule and reliance should not be placed upon the supposed immunity of the family because the tuberculosis is complicated by or consequent upon silicosis.

## Control and Prevention of Silicosis

The control of silicosis is both an engineering and a medical problem, as are so many other situations that concern the public health. Both the engineer and the physician, with their technicians, have their parts to play. In some instances, the medical phase may predominate; in others, the engineering, but it is seldom that they can be separated or be independent of each other if a thorough job of prevention is to be done.

### Engineering Control

The development of silicosis is dependent upon the dosage of silica and the matter of avoiding a dangerous quantity might be thought simple. The lay mind, familiar with the definiteness of the hazard of carbon monoxide, and other toxic substances producing acute effects, usually fails to appreciate the inherent difficulties of establishing a threshold limit for a substance producing a chronic disease whose development is measured in terms of years rather than hours.

From time to time, critical comment is directed toward the industrial hygienist by the engineer because the forme has failed to inform the latter of the safe or threshold limit o

silica dust in the air of working places. That is not an unnatural criticism from those whose profession involves dealing with definite and measurable factors. Unfortunately, neither physiology nor pathology in their relationship to the incidence and progress of chronic disease has yet advanced to the point where they are susceptible of expression in terms as definite as those used by the engineer.

The circumstances under which silica dust is introduced into the air of working places varies to an extreme degree. Furthermore, these circumstances and others that, in turn, affect them, vary from day to day and from hour to hour, they are affected by wind and weather, by dryness and humidity. All this would be complex enough if we were dealing only with the dust of pure silica, but in actual practice, the silica dust is frequently combined with other substances which may modify its action and about which little is known. Some industrial processes lend themselves to dust control measures; others do not, and in these latter, an entire change of the process itself may be necessary to overcome the hazard

## Dust Sampling

To deal with any health hazard successfully, its nature and severity must be understood. For silica dust, the two factors involved are the proportion of silica in the particular rock or mineral substance from which the dust originates and the amount of dust present in the air breathed by the workmen exposed. A great deal of study and research has been concentrated on this subject It is customary to define the extent of exposure by stating the number of dust particles, under ten microns in greatest diameter, in millions per cubic foot of air.

Various methods of counting the dust particles in the air have been devised, later to be discarded, until at the present time in the United States reliance is chiefly placed on the impinger method as defined and practiced by the United

States Public Health Service and the United States Bureau of Mines. The merit of the impinger is that it takes large samples, over a considerable period of time (one-half to one or two hours). The objection to the impinger is that it is cumbersome, time consuming, and demands expert technical skill if the results are to be trustworthy. Simpler devices consisting of smaller portable instruments, taking a small quantity of air — "grab samples" — and necessitating less skill are also used. The objection to these devices is that due to the smallness of the sample, the margin of error is considerable. Both methods have their place, but when serious studies of dust hazards are involved, it is probable that reliance will continue to be placed on the impinger method until a more satisfactory one is evolved. Small electric precipitators, modeled on the Cottrell precipitator, are in use but are mostly restricted to laboratory practice and to standardize the impinger. Other devices are still in the laboratory or experimental stage.

To summarize. While no one method of sampling air for the purpose of making dust counts has proved entirely satisfactory, up to now the impinger is accepted as most reliable in field work. No two methods are quite comparable, even samples taken by the same method in the same location and at the same time frequently show marked differences.

State Departments of Labor are naturally desirous of establishing standards of air dustiness for the control of the dust hazard and for the guidance of factory managers and factory inspectors. To a very limited extent, such standards have been suggested as a result of various government investigations.[28] However, much caution is needed here and too much reliance should not be placed upon the formulation of "standards" of air dustiness and the enactment of factory codes setting forth permissable limits of dust counts. Such threshold limits as have been worked out have very definite value as a guide in endeavoring to secure better working conditions but ther

limitations and possible transitory nature should be well understood. They should not as yet be enacted into law, otherwise a situation analogous to that of school ventilation may be repeated. Some of our State laws and regulations controlling ventilation in schools provide for an air change of 30 cubic feet per minute per occupant—a highly expensive and troublesome provision. This standard was based on what was thought at the time these laws were enacted to be a correct conception of "pure" air and "bad" air. Our present knowledge of ventilation and atmospheric control has proved the inadequacy of this carbon dioxide standard but it is the law in many places. If history repeats itself in formulating threshold limits of air dustiness, the result will be not only vexatious but costly.

The first and basic principle in dealing with a dust hazard is to remove the dust at its point of origin and thus prevent its dissemination into the air. This is the only procedure to which the term prevention may be properly applied. Once the dust is in the air, the protection of the individual becomes difficult and often unsatisfactory.

The removal of dust at the point of origin may be accomplished by suction devices working on the principle of the well known vacuum cleaner. In certain processes, such as drilling, water may be used to advantage. In some instances, water and air suction may be combined. So great is the variety of industrial processes from which silica dust may arise that a detailed description is not feasible. Each type of process usually presents its own problems and difficulties to the engineer and what will work satisfactorily under one set of circumstances will not always be successful in another.

Once the dust has been disseminated into the air, its removal may present serious engineering difficulties. Water may be employed, either as a spray to increase the humidity and facilitate the settling of dust particles, or water blasts may be

used to accomplish the same results more rapidly and thoroughly. It is obvious that this method is limited in its application. The dust may be diluted by increasing the quantity of air through forced ventilation — again a method not always applicable and apt to be expensive The two methods may be combined Both methods, either singly or in combination, have been widely applied in mining operations

Some experimentation has been done with the use of hygroscopic substances applied to the surfaces of mine passages to prevent dust from being continually stirred into the air. It has also been proposed to add to silica dust other kinds of dust with the hope of inhibiting the action of silica in the pulmonary tissues While this method of combatting silica dust may be both practical and valuable under certain conditions, it is not likely that it will be accepted as a final satisfactory solution. The effects of long continued breathing of such mixed dusts are unknown. Boiling or chlorinating infected drinking water may be a necessary procedure but satisfactory hygienic standards require that the community be supplied with water from reasonably pure sources and enormous sums of money are spent to secure such water Likewise the air that industrial workers must breathe should be reasonably pure Air containing harmful quantities of an injurious substance plus a counter-acting or antidotal substance would seem to leave much to be desired with respect to purity and proven harmlessness The industrial age has seen too many miracles of achievement and too many evidences of inventive genius to make one believe that satisfactory dust control is beyond our resources.

It is essential that where dust control devices have been installed that check tests be made from time to time to determine their continued efficiency Such equipment tends to lose its value unless continuously supervised and maintained in good working condition It is the common experience of

investigators to find installations of ventilating and dust removal machinery that are of little use due to failure to keep them in proper working order while at the same time they continue to give a feeling of unwarranted security.

Face masks, helmets, and respirators have been developed to protect the individual in dust polluted atmospheres Their use is justified when the exposure, or the dusty process itself, is intermittent and of short duration. Filter type respirators, which are approved by the United States Bureau of Mines as adequate for protection against silica dust, can be relied on. They may be uncomfortable, especially when the temperature is high, and their continued use calls for a degree of co operation from the individual workers which experience has shown is difficult to maintain Positive pressure helmets and masks are generally efficacious when used under conditions for which they are suited. The adequate supervision and maintenance of protective equipment is all important A faulty device or one improperly used will give a false sense of security that will eventually betray its user.

### Medical Control

Pre-employment physical examinations, where a definite health hazard is involved, are an essential public health procedure, definitely important in controlling the incidence of occupational disease. The benefit should accrue impartially to the individual, the industry, and the community It is also important that the function of the pre-employment examinations be understood so that they may not be abused It is desirable to place men in types of work for which they are adapted It is equally necessary not to bar, needlessly, employment to those seeking it Much of the dispute and ill feeling engendered during the last few years on the subject of physical examinations, both pre-employment and for those already employed, as related to the silicosis furor, might have been

avoided if the basic reasons for such examinations had been understood and if employers and others concerned had thought their problem through. Some misunderstanding was inevitable under the general circumstances.

It is not practicable to lay down definite rules or to assume a dogmatic attitude Circumstances vary in different occupations and different parts of the country and economic considerations always stand well to the fore and must be recognized.

Obviously no one with active or recently active tuberculosis should be exposed to silica dust, nor anyone suffering from other pulmonary disease, especially if of an infective nature, which would tend to increase the hazard On the other hand, there is often no good reason for rejecting an applicant who already has silicosis, if the silica hazard in the work place involved is under control and if the workman's physical condition is properly supervised thereafter This is an important consideration where the industry is a dominant one in its locality and other opportunities for work are limited Here is where the experienced industrial physician must bring his judgment to bear both upon the individual and upon the work he is to do

In connection with the recent compensation laws passed in some of the States, the attitude toward pre-employment examinations is interesting. The New York [5] law states, "It is hereby declared to be the policy of the legislature of this State, in enacting this article, to prohibit, through every lawful means available, any requirement to employment which compels an applicant for employment in any occupation coming within the purview of this article to undergo a medical examination "

As contrasted to this, the North Carolina [7] law states, (where the Industrial Commission has found an occupation to have a hazard of asbestosis or silicosis) "it shall be the

duty of every employer . . . to provide prior to employment necessary examinations of all new employees for the purpose of ascertaining if any of them are in any degree affected by asbestosis and/or silicosis or peculiarly susceptible thereto, and every such *employer shall from time to time, as ordered* by the Industrial Commission, provide similar examinations for all of his employees whose employment exposed them to the hazards of asbestosis and/or silicosis" At least one member of the Advisory Medical Committee or other physician designated by the Industrial Commission shall make such examination or be present when such examination is made. Further on, the law continues: "The refusal of an employee to submit to any such examination shall bar such employee from compensation or other benefits provided by this Act in the event of disablement and/or death resulting from an exposure to the hazards of asbestosis and/or silicosis subsequent to such refusal."

Particularly to be commended are the control methods set up in the Province of Ontario A co-operative agreement is in force *between the Workmen's Compensation Board and the* Provincial Health Department. Examination centers are established where necessary and pre-employment and periodic physical examinations, with the use of the roentgen ray are made by a physician of the Health Department for the Workmen's Compensation Board Examination certificates are issued to acceptable workers Claimants for compensation come before a Referee Board of Physicians of the Health Department. This Board works in co-operation with the industries concerned and the Compensation Board.

Where workmen are exposed to a silica hazard, medical supervision goes hand in hand with engineering control of the dust Indeed, it is the industrial physician who must finally determine whether the dust control measures in his establishment are *efficacious Annual, or perhaps bi-annual,*

medical examinations with the roentgen-ray are usually sufficient with more frequent examinations of those individuals who, the industrial physician feels, need more constant supervision. As in any other circumstance where an occupational health hazard is involved, no mechanical method of prevention can be taken for granted.

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# INDEX